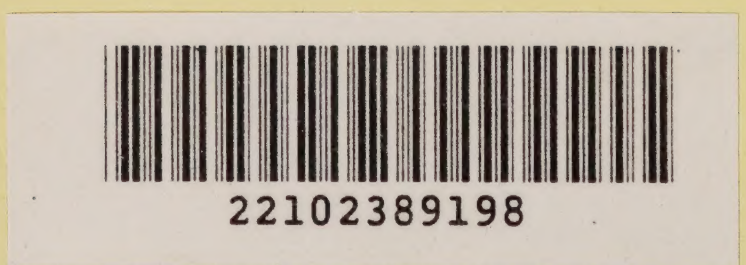
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OF

THE MEDICAL SOCIETY

OF

LONDON.

VOLUME THE TWENTY-SECOND.

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WILLIAM H. BATTLE, F.R.C.S.,

AND

JAMES CALVERT, M.D., F.R.C.P.

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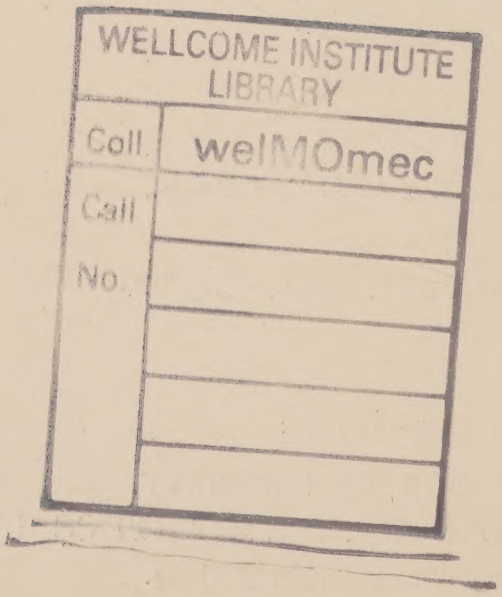
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1859. Charles Henry Felix Routh, M.D.

1860. John Gay.
1861. Arthur Leared, M.D.
1862. Victor de Méric.
1863. Samuel Osborne Habershon, M.D.
1864. John Louis William Thudichum, M.D.
1865. Robert Greenhalgh, M.D.
1866. Thomas Christopher Weeden Cooke.
1867. Frederick William Headland, M.D.
1868. William Frederick Teevan.
1869. George Duncan Gibb, M.D.
1870. Francis Mason.
1871. William Cholmeley, M.D.
1872. Frederick James Gant.
1873. John Cockle, M.D.
1874. Robert Brudenell Carter.
1875. George Buchanan, M.D.
1876. Erasmus Wilson, F.R.S.
1877. John Hughlings Jackson, M.D., F.R.S.
1878. Alfred Carpenter, M.D.
1879. Walter John Coulson.
1880. William Henry Broadbent, M.D.
1881. Arthur Edward Durham.
1882. Edmund Symes Thompson, M.D.
1883. Edward Lund.
1884. Charles Theodore Williams, M.D.
1885. George Murray Humphry, M.D., F.R.S.
1886. Richard Douglas Powell, M.D.
1887. Sir William MacCormac.
1888. Sir Joseph Fayrer, K.C.S.I., M.D., F.R.S.
1889. Jonathan Hutchinson, F.R.S.
1890. Arthur Ernest Sansom, M.D.
1891. Sir Joseph Lister, Bart., F.R.S.
1892. Sir James Crichton-Browne, M.D., F.R.S.
1893. W. Mitchell Banks.
1894. William M. Ord, M.D.
1895. A. Pearce Gould.
1896. William H. Allchin, M.D.
1897. Edmund Owen.
1898. Frederick T. Roberts, M.D.
1899. Alban Doran.

THE FOTHERGILLIAN GOLD MEDALLISTS.

1787. William Falconer, M.D.
1790. Robert Willan, M.D.
1791. John Coakley Lettsom, M.D.
1795. John Mason Good.
1801. Francis Bouttatz, M.D.
1803. Edward Jenner, M.D.
1824. Robert W. Bampfield.
1828. John George Parry.
1831. William Augustus Guy.
1834. William James Clement.
1835. George Moore.
1836. Thomas Egerton Bryant.
1838. George Pilcher.
1840. Samuel Osborn.
1842. James Risdon Bennett, M.D.
1843. John Weaver Lever, M.D.
1844. Henry Pratt Robarts.
1845. Walter Cooper Dendy.
1846. Robert Mortimer Glover, M.D.
1847. Silas Stedman.
1849. John Milligan.
1850. Richard Payne Cotton, M.D.
1851. Richard Hodges.
1852. Frederick William Headland.
1853. Alfred William Poland.
1854. Benjamin Ward Richardson, M.D.
1856. William Burke Ryan.
1857. Edwin Canton.
1858. Thomas Herbert Barker, M.D.
1859. Alderman Thomas Houghton Waters.
1868. John Clay.
1870. Thos. Smith Clouston, M.D.
1872. Edwards Crisp, M.D.
1873. John Kent Spender, M.D.
1877. Peter Murray Braidwood, M.D.
1878. John Milner Fothergill, M.D.
1882. Thomas Michael Dolan, M.D.
1883. Norman Porritt.
1886. John Strahan.
1888. Hobart Amory Hare, M.D., U.S.A.
1893.* William Richard Gowers, M.D., F.R.S.
1896. Victor Horsley, F.R.S.
1899. Sydney Monckton Copeman, M.D.

* *First Triennial Award under the Charity Commissioners' New Scheme,* vide *Trans., vol. xiv, p. xi.*

THE HONORARY FELLOWS.

1893. Barnes, Robert, M.D., Bernersmede, Eastbourne, LL, C.

1896. Berger, Paul, 16, Rue de Bourgogne, Paris. Professor of Clinical Surgery in the Faculty of Medicine, Member of the Academy of Medicine, Surgeon to "l'hôpital de la Pitié,"

1897. Bergmann, Prof. E. von, M.D., Berlin.

1881. Billings, John S., M.D., Washington, Surgeon to the United States Army.

1899. Brouardel, Paul Camille Hippolyte, 85, Boulevard, St. Germains, Paris. Professor of Medical Jurisprudence, Dean of the Faculty of Medicine, University of Paris; Member of the Institute and of the Academy of Medicine.

1894. Catrin, Louis, M.D., Médecin-major de première classe, Professeur agrégé à l'Ecole du Val-de-Grace, Paris.

1873. Chauveau, A., late Professor of Physiology at the Medical School of Lyons, 10, Avenue Jules Janin, Paris.

1890. Crudeli, Tommasi, M.D., Rome.

1881. Da Costa, J. M., M.D., Professor of Medicine in the Jeffreson Medical College, 1700, Walnut-street, Philadelphia.

1881. Emmet, Thomas Addis, M.D., 89, Madison-avenue, Surgeon to the Woman's Hospital of the State of New York.

1886. Gairdner, Sir William Tennant, K.C.B., M.D., LL.D. Edin., F.R.C.P. Edin., F.R.S., 225, St. Vincent-street, Glasgow.

1894. Gant, Frederick James, F.R.C.S., 16, Connaught-square, W., Consulting Surgeon to the Royal Free Hospital. P, VP 2, LL, O, C 3.

1897. Guyon, Felix, M.D., Professor in the Faculty of Medicine, Member of the Institute and of the Academy of Medicine, Paris.

1897. Hingston, Sir W. Hales, M.D., Montreal.

1890. Kocher, Theodor, Professor, Berne.

1883. Le Roy de Mericourt, A., M.D., Paris.

1878. Mitchell, S. Weir, M.D., Walnut-street, Philadelphia.

1875. Ollier, Professor, Lyons.

1899. Osler, William, M.D., Baltimore, U.S.A., Professor of Medicine, John Hopkins University, and Physician-in-Chief, John Hopkins Hospital.

1899. Pachoutin, Victor Vasilivitch, 6, Nijni Grodskia, St. Petersburg; President of the Imperial Military Medical Academy and of the Medical Council.

1873. Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., 5, Park-square, W., Serjeant-Surgeon to H.M. the Queen; Surgeon to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital.

1899. Potain, Carl Edouard, 250, Boulevard St. Germains, Paris; Professor to the Faculty of Medicine, University of Paris; Member of the Academy of Medicine and of the Academy of Science; Physician to "l'hôpital de la Charité."

1899. Rauchfuss, Charles Andreivitch, 8, Ligovka, St. Petersburg, Director of the Prince of Oldenburg's Hospital for Children.

1897. Roddick, T. G., M.D., 80, Union-avenue, Montreal.

1877. Sanné, A., 33, Rue de Naples, Paris.

1899. Sklifosoffsky, Nicholas Vasilivitch, M.D., Director and Professor of Surgery, Imperial Clinical Institute of the Princess Helena, Paulovna, St. Petersburg.

1873. Virchow, Rudolph, M.D., Professor of Pathological Anatomy in the University of Berlin.

CORRESPONDING FELLOWS.

1882. Badaloni, Giuseppe, M.D., Bologna, Italy.
1856. Baker, Albert, M.D., Clarence House, Exmouth.
1855. Beardsley, Amos, Bay Villa, Grange, Lancashire.
1850. Beneke, F. W., M.D., New York.
1850. Böhm, Professor, M.D., Vienna.
Bottini, Enrico, M.D., Milan.
1855. Coates, Charles, M.D., F.R.C.P., 10, Circus, Bath, Consulting Physician to the Bath Royal United Hospital. c 3.
1850. Cox, William Isidore, Hawkesbury-Upton, Gloucestershire. c.
1853. Fallot, R., M.D., St. Laurent d'Aigonze, Montpelier, France.
1889. Frank, Philip, M.D., F.R.C.P., Cannes, France.
1876. Griffith, Richard Glyn, Allahabad, India.
1896. Hamilton, Allan McLane, M.D., 44, East Twenty-ninth Street, New York.
1864. Hasenfeld, Emmanuel, M.D., Pesth.
1875. Jones, Philip Sydney, M.D., F.R.C.S., Examiner in Medicine in the University of Sydney, Australia, Hon. Consulting Surgeon to the Sydney Infirmary.
1851. Kölliker, Albert, M.D., Professor of Anatomy and Physiology at the University of Wurzburg.
1876. Leighton, Walter H., M.D., Lowell, Massachusetts, U.S.A.
1851. Negri, Gaetano, M.D. Pisa.
1865. Peruzzi, Domenico, M.D., 22, Via Mazzini, Bologna.
1886. Rocha, A., M.D., Professor of Clinical Medicine at the University of Coimbra, Beira, Portugal.
1860. Roussel, M.D., Dean of the Faculty of Medicine, Montpelier.
Scharlan, Gus. W., M.D., Stettin, Prussia.
1876. Schmitz, Richard, M.D., Neuenahr.
1874. Schutgowsky, J., St. Petersburg.
1851. Stockwell, Thomas Goldesbrough, F.R.C.S., 6 Circus, Bath, Surgeon to the Bath Royal United Hospital.
Williams, Charles, F.R.C.S. Edin., 48, Prince of Wales-road Norwich; Senior Surgeon to the Norfolk and Norwich Hospital.

THE FELLOWS

OF

THE MEDICAL SOCIETY OF LONDON.

(Corrected to 1st November, 1899.)

Explanation of Abbreviations.

P.—President.
VP.—Vice-President.
T.—Treasurer.
L.—Librarian.
S.—Secretary.
C.—Councillor.
LL.—Lettsomian Lecturer.
FM.—Fothergillian Gold Medallist.
SM.—Silver Medallist.
O.—Orator.
CFC.—Chairman, House and Finance Committee.
§—Sec. for Foreign Correspondence.
*—Life Members.
Tr.—Trustee.

The number prefixed signifies the date of election. The figures appended indicate the number of Sessions served, and refer to past appointments ONLY.

1890. Abbot-Anderson, William Maurice, M.B., 37, Wimpole-street, W.
1888. Abbott, Charles Edward, M.R.C.S., 1, Wellington-place, Cheltenham.
1891. Abraham, Phineas S., M.D., 2, Henrietta-street, Cavendish-square, W.
1896. Abrahams, Bertram Lewis, M.B., 14, Welbeck-street, W.
1894. Achard, Alexander Louis, M.D., 9, Blandford-street, Portman-square, W.
1890. Ackland, Robert Craig, M.R.C.S., 54, Brook-street, Grosvenor-square, W.
1883. Acland, Theodore Dyke, M.D., 74, Brook-street, Grosvenor-square, W. c 2.
1884. Adam, James, M.D., Malling-place, West Malling, Kent.
1878. Adams, Josiah Oake, M.D., Brook House, Upper Clapton, E.
1852. *Adams, William, F.R.C.S., 7, Loudoun-road, St. John's-wood, N.W. p, c 8, o, vp 3, ll.
1878. *Allchin, William Henry, M.D., F.R.S.E., 5, Chandos-street, Cavendish-square, W. sm, l 18, vp 2, o. *Councillor.*
1873. Allen, Henry Marcus, F.R.C.P. Edin., 17, Palmeira-square, Brighton.
1873. Allfrey, Charles Henry, M.D., Plas Newydd, St. Leonards-on-Sea.

1883. Allingham, Herbert W., F.R.C.S., 25, Grosvenor-street, W. c 3.
1872. *Allingham, William, F.R.C.S., 25, Grosvenor-street, W. c.
1894. Alston, William Evelyn, M.B., Wheathampstead, Herts.
1860. Althaus, Julius, M.D., 26, Queen Anne-street, W. c 5, § 3.
1885. Anderson, John, M.D., C.I.E., 9, Harley-street, W. c 3.
1889. Anderson, William, F.R.C.S., 5, Cavendish-square, W.
1888. Andrewes, Frederick William, M.B., Highwood, Hampstead-lane, Highgate, N.
1869. Armitage, Samuel Harris Tatham, M.D., 39, Grosvenor-street, W.
1894. Armstrong, William, M.R.C.S., J.P., Thorncliffe, Buxton.
1894. Ashe, William Percy, M.D., 17, Alexander-square, S.W.
1873. Atkinson, Edward, M.R.C.S., 93, Albion-street, Leeds.
1892. Ayres, Charles James, M.D., 55a, Welbeck-street, W.

1873. Bagshawe, Frederic, M.D., 35, Warrior-square, St. Leonards-on-Sea. c.
1892. Bailey, Henry Frederick, M.R.C.S., The Hollies, Lee-terrace, Lee, S.E. c 2.
1894. Bailey, Robert Cozens, M.S., 21, Welbeck-street, W.
1891. Baily, Percy J., M.B., County Asylum, Hanwell, W.
1898. Bain, William, M.D., Shaythorpe, York-place, Harrogate.
1876. *Baker, Henry Francis, F.R.C.S. Edin., 2, Mandeville-place, Manchester-square, W. c.
1890. Baker, William Henry, M.R.C.S., 152, Westbourne-grove, W. c. *Councillor.*
1891. Ball, James Barry, M.D., 12, Upper Wimpole-street, W.
1881. Ballance, Charles Alfred, M.S., 106, Harley-street, W. s 2, c 3.
1884. Banks, Sir W. Mitchell, F.R.C.S., LL.D., 28, Rodney-street, Liverpool. o, c. *Lettsomian Lecturer.*
1898. Barnard, Harold Leslie, M.S., 9, Upper Wimpole-street, W.
1883. *Barnes, Robert, M.D., *Honorary Fellow* (*q. v.*).
1874. Barrett, Howard, M.R.C.S., 49, Gordon-square, W.C. c 3. *Vice-President.*
1896. Barrett, Walter Russell, M.R.C.S., 6, Chandos-street, Cavendish-square, W.
1884. Barrow, Albert Boyce, F.R.C.S., 37, Wimpole-street, W. c.
1886. Barwell, Richard, F.R.C.S., 55, Wimpole-street, W.
1884. Bateman, Frederick Augustus Newton, M.R.C.S., 4, Charles-street, St. James's-street, S.W.
1886. Batterham, John Williams, M.B., Bank House, Grand-parade, St. Leonards-on-Sea.
1888. Battle, William Henry, F.R.C.S., 49, Harley-street, W. c. s 2. *Councillor.*
1896. Bayliss, Richard Arthur, M.R.C.S., 26, Gay-street, Bath.
1882. Beach, Fletcher, M.B., Winchester House, Kingston-hill, Surrey, and 64, Welbeck-street, W. c.

1887. Beale, Edwin Clifford, M.B., 23, Upper Berkeley-street, W. c 2.
1891. Beale, Peyton T. B., F.R.C.S., 61, Grosvenor-street, W.
1880. Beevor, Charles Edward, M.D., 33, Harley-street, W. s 2, c. *Trustee.*
1889. Beevor, Sir Hugh Reeve, Bart., M.D., 17, Wimpole-street, W.
1881. Bennet, Robert Ottiwell-Gifford, M.D., Tankerville House, Park-place, Buxton.
1883. Bennett, William Henry, F.R.C.S., 1, Chesterfield-street, Mayfair, W. c.
1896. Berkeley, Comyns, M.B., 53, Wimpole-street, W.
1887. Berry, James, F.R.C.S., 21, Wimpole-street, W.
1898. Best, William Harris, L.S.A., Ilford, Essex.
1873. Beveridge, James Spowart, M.R.C.P. Edin., Foulsham, Norfolk.
1890. Bidwell, Leonard Arthur, F.R.C.S., 59, Wimpole-street, W.
1868. Bird, George, M.D., 6, Windmill-hill, Hampstead, N.W.
1888. Bird, Matthew Mitchell, M.D., St. Mary's Hospital, W.
1850. *Birkett, John, F.R.C.S., 1, Sussex-gardens, W. vp, c 6.
1895. Bishop, Edward Stanmore, F.R.C.S., 316, Oxford-road, Manchester.
1889. Bisshopp, Francis Robert Bryant, M.B., Belvedere, Lonsdale-gardens, Tunbridge Wells.
1886. *Black, William Gall, F.R.C.S., 2, George-square, Edinburgh.
1897. Blacker, George Francis, M.D., 11, Wimpole-street, W.
1881. Blaker, Walter Campbell, M.R.C.S., 4, Denmark-villas, Hove, Sussex.
1888. Blanc, Leon, M.D., Aix les Bains, France.
1871. *Bloxam, John Astley, F.R.C.S., 75, Grosvenor-street, W. vp 2, s 2, c 3.
1867. Bond, Thomas, F.R.C.S., 7, The Sanctuary, Westminster, S.W. c.
1879. Bott, Henry, M.R.C.S., Brentford, Middlesex.
1886. Bourns, Newcome Whitelaw, M.D., 78, Redcliffe-gardens, S.W.
1886. Boustead, Robinson, M.D., Lieutenant-Colonel, R.A.M.C., c/o Messrs. King & Co., 45, Pall Mall, S.W.
1895. Bower, David, M.D., Springfield House, Bedford.
1889. Bowles, Robert Leamon, M.D., 16, Upper Brook-street, W. c 3.
1895. Boyd, Stanley, F.R.C.S., 134, Harley-street, W. c. *Councillor.*
1899. Bradford, J. Rose, M.D., F.R.S., 60, Wimpole-street, W.
1896. Bradley, James Edward Campbell, M.B., Greville House, Raynes Park, Wimbledon.
1889. Braine, C. Carter, F.R.C.S., 26, Wimpole-street, W.
1876. Brewer, Alexander Hampton, M.R.C.S., 136, Richmond-road, Dalston, N.E.
1896. Bridges, E. Chittenden, M.B., Priory-mansions, Drayton-gardens, South Kensington, S.W.
1873. Bridgwater, Thomas, M.B., LL.D., J.P., Harrow, Middlesex.
1893. Broadbent, John Francis Harpin, M.B., 35, Seymour-street, W.
1862. Broadbent, Sir William Henry, Bart., M.D., F.R.S., 84, Brook-street, W. p, vp, o, ll, c 4.

1890. Brook, William Frederick, F.R.C.S., Longlands House, Swansea.
1898. Brooke, Gilbert E., L.R.C.P., East Harbour, Turk's Island, West Indies.
1878. Brown, Andrew, M.D., 27, Lancaster-road, Belsize-park, N.W.
1889. Browne, George Buckston, M.R.C.S., 80, Wimpole-street, W. c 2.
1873. Browne, Lennox, F.R.C.S. Edin., 15, Mansfield-street, Portland-place, W.
1887. Bruce, John Mitchell, M.D., 23, Harley-street, W. c 2. *Vice-President.*
1873. Brunjes, Martin, M.R.C.S., 39, Blenheim-gardens, Willesden-green, N.W.
1898. Brunton, John, M.B., 16, Endsleigh-street, Tavistock-square, W.C.
1874. *Brunton, Thomas Lauder, M.D., F.R.S., 10, Stratford-place, W. ll, vp, c 4, sm.
1850. *Bryant, Thomas, F.R.C.S., 65, Grosvenor-street, W. p, vp, ll, s 2, c 4. *Trustee.*
1897. Bunch, John Le Mare, M.D., 32, Wimpole-street, W.
1885. *Bunny, J. Brice, M.R.C.S., Warre House, Bishops Lydeard, Taunton.
1886. Butler-Smythe, Albert Charles, F.R.C.S., 76, Brook-street, W.
1872. Byas, Edward Hegley, M.R.C.S., 10, Cambridge-gate, Regent's Park, N.W.

1886. Cahill, John, M.D., 12, Seville-street, Lowndes-square, Hyde Park, S.W. c 3.
1892. Caldwell, Robert, F.R.C.S., Major, R.A.M.C., care of Messrs. Holt and Co., 17, Whitehall-place, S.W.
1893. Caley, Henry Albert, M.D., 24, Upper Berkeley-street, W. *Councillor.*
1891. Calvert, James, M.D., St. Bartholomew's Hospital, E.C. *Hon. Secretary.*
1897. Cameron, Ewan Gordon, M.R.C.S., 64, Great Portland-street, W.
1892. Cargill, Lionel Vernon, F.R.C.S., 15, Stratford-place, W.
1892. Carless, Albert, F.R.C.S., 10, Welbeck-street, W.
1882. Carpenter, Arthur Bristowe, M.B., Wykeham House, Bedford Park, Croydon.
1889. Carr, John Walter, M.D., 19, Cavendish-place, W.
1897. Carré, L. J. Gerard, M.D., 27th Baluchis, Uganda, British East Africa.
1871. Carter, Robert Brudenell, F.R.C.S., 31, Harley-street, W. p, vp, o, ll, c 4.
1889. Cartwright, Alexander, M.R.C.S., 32, Old Burlington-street, W.
1876. Cartwright, S. Hamilton, *Travelling.*
1876. *Cathcart, Samuel, M.R.C.P. Edin., 11, Hornsey-lane-gardens, Highgate, N.
1889. Cautley, Edmund, M.D., 15, Upper Brook-street, W.
1896. Chapman, Charles William, M.D., 21, Weymouth-street, W.

1885. Chasseaud, William, M.D., Smyrna, Asia Minor.
1889. Cheyne, Watson, F.R.C.S., F.R.S., 75, Harley-street, W. ll, c.
1877. *Chisholm, Edwin, M.D., 82, Darlinghurst-road, Sydney, New South Wales.
1895. Christmas, Robert William Samuel, M.R.C.S., 23, Hopton-road, Streatham, S.W.
1871. Churton, Thomas, M.D., 35, Park-square, Leeds. c.
1875. Clark, Andrew, F.R.C.S., 71, Harley-street, W. c.
1883. Clarke, William Bruce, F.R.C.S., 51, Harley-street, W. c.
1894. Clinch, Thomas Aldous, M.D., County Asylum, Winterton, Ferryhill, co. Durham.
1879. *Clutton, Henry Hugh, F.R.C.S., 2, Portland-place, W. vp 2, c 3.
1899. Cobb, Ernest Herbert, M.R.C.S., Victoria Hospital, Folkestone.
1894. Cochrane, James Mackeand, M.D., 10, Weymouth-street, W.
1849. *Cockle, John, M.D., The Lodge, West Molesey, Surrey. p, vp, o, l 3, c 3, sm.
1899. Cohen, George Alexander, M.B., 17, Grand-parade, Harringay, N.
1893. Cole, Robert Henry, M.D., Moorcroft, Hillingdon, Uxbridge.
1887. Collier, William, M.D., St. Mary's Entry, Oxford. c.
1892. Colman, Walter S., M.D., 22, Wimpole-street, W. *Councillor.*
1871. Cook, John, M.D., 1, Nottingham-terrace, Regent's Park, N.W.
1862. Cooper, Alfred, F.R.C.S., 9, Henrietta-street, Cavendish-square, W. c 3, vp.
1888. Cooper, Arthur, M.R.C.S., 20, Old Burlington-street, W.
1894. Cooper, Peter, M.R.C.S., 35, Shooters-hill-road, Blackheath, S.E.
1894. Corbould, Victor A. L. E., M.D., 43, Victoria-road, Kensington, W.
1872. Corfield, William Henry, M.D., 19, Savile-row, W. c.
1899. Corner, Harry, M.D., Brook House, Southgate, N.
1898. Cotton, Holland John, M.D., 2, Eccleston-houses, St. George's-road, S.W.
1891. Coumbe, John Batten, F.R.C.S., 55, High-street, Lowestoft.
1879. Coupland, Sidney, M.D., 16, Queen Anne-street, W. vp 2, c 3.
1889. Courtney, Guy Budd, M.B., 28, Augusta-road, Ramsgate.
1873. Craven, Sir Robert Martin, F.R.C.S., J.P., 14, Albion-street, Hull.
1889. Crawford, James, M.D., Grosvenor-mansions, 80, Victoria-street, S.W.
1899. Creasy, L. Eliot, M.R.C.S., 35a, Welbeck-street, W.
1871. Crichton-Browne, Sir James, M.D., F.R.S., 61, Carlisle-place-mansions, S.W. p, o, c 2.
1881. Cripps, William Harrison, F.R.C.S., 2, Stratford-place, W. c 2.
1880. Critchett, George Anderson, F.R.C.S. Edin., 21, Harley-street, W.
1880. Crocker, Henry Radcliffe, M.D., 121, Harley-street. c 3.
1881. Cross, Francis Richardson, F.R.C.S., Worcester House, Clifton, Bristol.
1890. Cullingworth, Charles James, M.D., 14, Manchester-square, W. c 3.
1874. Cumberbatch, Alphonso Elkin, F.R.C.S., 80, Portland-place, W. c 2.

1897. Dakin, William Radford, M.D., 18, Grosvenor-street, W.
1894. Dalby, John Lyttleton, M.R.C.S., 13, Buckingham-road, Brighton.
1871. Dalby, Sir William Bartlett, F.R.C.S., 18, Savile-row, W. p, c 2.
1881. Dallaway, Dennis Joseph William, L.R.C.P. Edin., Piccadilly Club, W. *Councillor.*
1890. Davis, Henry, M.R.C.S., 60, Queen Anne-street, W.
1889. *Davison, James, M.D., "Streate Place," Bath-road, Bournemouth.
1897. Davison, Rashell, M.D., Vernham, New Malden, Surrey.
1880. Davson, Smith Houston, M.D., Campden Villa, 203, Maida-vale, W. c 3.
1868. *Davy, Richard, F.R.C.S., F.R.S.E., Burstone House, Bow, North Devon. vp, s 2, sm, § 2.
1876. Dawes, Richard St. Mark, M.R.C.S., Gawler, South Australia.
1897. Dawson, Bertrand, M.D., 110, Harley-street, W.
1880. Dawson, Yelverton, M.D., Heathland, Southbourne-on-Sea, Christchurch, Hants.
1894. Dean, Henry Percy, M.S., 69, Harley-street, W.
1883. Dent, Clinton Thomas, F.R.C.S., 61, Brook-street, Grosvenor-square, W. c 3.
1897. Des Vœux, Harold A., M.D., 8, James-street, Buckingham-gate, S.W.
1891. Diver, Ebenezer, M.D., "Garstone," St. Peter's-road, St. Margaret's, Twickenham.
1893. Dockrell, Morgan, M.D., 9, Cavendish-square, W.
1885. Dodd, Henry Work, F.R.C.S., 136, Harley-street, W.
1882. Dolan, Thomas Michael, M.D., Horton House, Halifax. fm 1882.
1881. Doran, Alban Henry Griffiths, F.R.C.S., 9, Granville-place, W. c 3, o, § 3. *Councillor.*
1898. Douglas, John L., M.D., 42, Central-hill, Norwood, S.E.
1890. Douglas, William, M.D., 4, St. Alban's-place, Regent-street, S.W.
1871. Dowse, Thomas Stretch, M.D., 14, Welbeck-street, W. § 3, c 3.
1897. *Drage, Lovell, M.D., Hatfield, Herts.
1886. Duckworth, Sir Dyce, M.D., 11, Grafton-street, Piccadilly, W. c.
1884. Duncan, William, M.D., 6, Harley-street, W.
1884. Durham, Frederick, F.R.C.S., 82, Brook-street, W.

1895. Ealand, Hugh Fawsit, L.R.C.P. Edin., The Moat, Puckeridge, Herts.
1896. Earle, Walter George, M.R.C.S., 44, Upper Grosvenor-road, Tunbridge Wells.
1891. Eastes, Thomas, M.D., 3, Shakespeare-terrace, Folkestone.
1893. Eccles, Arthur Symons, M.B., 23, Hertford-street, Mayfair, W.
1895. Eccles, William McAdam, M.S., 124, Harley-street, W.
1892. Eddowes, Alfred, M.D., 28, Wimpole-street, W.
1860. *Edmunds, James, M.D., 26, Manchester-square, W.
1880. Edwards, Frederick Swinford, F.R.C.S., 55, Harley-street, W. c 3.
1897. Elgood, Charles Reginald, M.D., 8, Clarence-villas, Windsor.

1868. Elliott, George Frederick, M.D., 1, Albion-street, Hull.
1882. Elliott, Thomas, M.D., Monson-place, Tunbridge Wells.
1889. Embleton, Dennis Cawood, M.D., St. Wilfrid's, St. Michael's-road, Bournemouth.
1883. English, Edgar, M.R.C.S., St. Mary's-road, Doncaster.
1880. English, Thomas Johnston, M.D., 128, Fulham-road, S.W.
1889. Esler, Robert, M.D., 4, Queen's-road, Peckham, S.E.
1894. Evans, Willmott Henderson, F.R.C.S., 13, Taviton-street, Gordon-square, W.C.
1893. Eve, Frederic S., F.R.C.S., 125, Harley-street, W.
1883. Ewart, Sir Joseph, M.D., J.P., Dep. Surgeon-General, Bengal Army (*retired*), Bewcastle, Dyke-road, Brighton. c.
1877. Ewart, William, M.D., 33, Curzon-street, Mayfair, W. c 3. *Hon. Sec. Foreign Correspondence.*

1884. Fardon, Edward Ashby, M.R.C.S., Middlesex Hospital, W.
1873. Fayrer, Sir Joseph, Bart., K.C.S.I., LL.D., M.D., F.R.S., 16, Devonshire-street, Portland-place, W. p, vp, ll, sm, o, c.
1896. Fayrer, Joseph, M.D., Captain, R.A.M.C., India.
1898. Felkin, Robert William, M.D., 23, Henrietta-street, Cavendish-square, W.
1884. Fenton, Frederick Enos, F.R.C.S., 33, Welbeck-street, W.
1888. Fenwick, Bedford, M.D., 20, Upper Wimpole-street, W.
1885. Fenwick, Edwin Hurry, F.R.C.S., 14, Savile-row, W. c.
1887. Ferrier, David, M.D. Edin., F.R.S., 34, Cavendish-square, W. c.
1878. Field, George, M.R.C.S., 34, Wimpole-street, W. c.
1894. Fisher, Frederick Charles, F.R.C.S., King's Langley, Herts.
1876. Fisher, Frederic Richard, F.R.C.S., 18, Wimpole-street, W.
1884. Flint, Arthur, M.D., J.P., Westgate Lodge, Westgate-on-Sea.
1878. *Fonmartin, Henry de, M.D., 26, Newberry-terrace, Lower Bullar-street, Southampton.
1893. Forbes, Norman Hay, F.R.C.S. Edin., Drumminor, Tunbridge Wells.
1879. Fowler, James Kingston, M.D., 35, Clarges-street, Mayfair, W. s 2, c, vp 2. *Councillor. Orator.*
1873. Fox, Arthur Edward Wellington, M.B., C.M., 16, Gay-street, Bath. c.
1887. Fox, Fortescue, M.D., Strathpeffer Spa, Ross-shire.
1871. Fox, Francis, M.R.C.S., 15, Arundel-gardens, Kensington Park, W. c 3.
1885. Fox, R. Hingston, M.D., 23, Finsbury-square, E.C.
1879. Fox, Thomas Colcott, M.B., 14, Harley-street, W. s 2, c 2.
1868. Freer, Alfred, M.R.C.S., J.P., Stourbridge, Worcestershire.
1896. Freyer, P. Johnston, M.Ch., Surgeon-Lieut.-Colonel (*retired*), 46, Harley-street, W.
1886. Frith, Baptist Gamble, M.B., 29, Cornwallis-gardens, Hastings.
1884. Frost, William Adams, F.R.C.S., 30, Cavendish-square, W. c 2.

1895. Galloway, James, M.D., 54, Harley-street, W.
1862. *Gant, Frederick James, F.R.C.S., *Honorary Fellow* (*q. v.*).
1847. *Garrod, Sir Alfred Baring, M.D., F.R.S., 10, Harley-street, W. P, VP 2, LL, O, C 9.
1887. Garrod, Archibald Edward, M.D., 9, Chandos-street, Cavendish-square, W. C 3.
1893. Garson, John George, M.D., 64, Harley-street, W.
1891. Gaster, Aughel, M.D., 68, Greencroft-gardens, South Hampstead, N.W.
1887. Gay, John, M.R.C.S., 119, Upper Richmond-road, Putney, S.W.
1856. *Gibbon, Septimus, M.B., 39, Oxford-terrace, W.
1882. Gibbons, Robert Alexander, M.D., 29, Cadogan-place, S.W.
1893. Gibson, Charles, M.D., Vanderbilt-court, Harrogate.
1897. Gibson, George Alexander, M.D., 17, Alva-street, Edinburgh.
1881. Giffard, Douglas W., M.R.C.S., 5, Pavilion-parade, Brighton.
1867. Gill, William, M.R.C.S., 11, Russell-square, W.C. C.
1894. Gill, Richard, F.R.C.S., 17, Wigmore-street, W.
1869. *Godson, Clement, M.D., 9, Grosvenor-street, W. VP, C 3, S 2, SM.
1873. Goodsall, David Henry, F.R.C.S., 17, Devonshire-place, Portland-place, W. VP 2. C, S 2, SM, CFC 7. *Treasurer; Chairman, House and Finance Committee.*
1878. *Gould, Alfred Pearce, M.S., 10, Queen Anne-street, W. VP 2, S 2, C 4, O.
1896. Gow, William John, M.D., 27, Weymouth-street, W.
1876. Gowers, Sir William Richard, M.D., F.R.S., 50, Queen Anne-street, W. VP, C, SM, LL. FM. 1893.
1887. Grant, James Edward Roney, M.R.C.S., 2, Charing Cross-chambers, Duke-street, Adelphi, W.C.
1896. Grant, J. Dundas, M.D., 8, Upper Wimpole-street, W.
1881. Green, Thomas Henry, M.D., 74, Wimpole-street, W. C 2. VP.
1886. Greves, Edwin Hyla, M.D., Rodney House, Bournemouth.
1873. Grieve, the Hon. Robert, M.D., C.M.G., British Guiana.
1885. Griffiths, Charles Thomas, L.R.C.P., 125, Soho-hill, Birmingham.
1884. Griffiths, Herbert Tyrrell, M.B., 5, Kensington-square, W.
1880. Gristock, William, M.D. Lond., 101, London-road, W. Croydon.
1893. Gubb, Alfred Samuel, M.D. Paris, 29, Gower-street, W.C.
1891. Guthrie, Leonard G., M.D., 15, Upper Berkeley-street, W.

1886. Habershon, S. Herbert, M.D., 70, Brook-street, Grosvenor-square, W.
1891. Hadley, Wilfred J., M.D., 58, Harley-street, W.
1887. Haig, Alexander, M.D., 7, Brook-street, W.
1881. Hall, Charles Ross, M.R.C.S., Hatfield, Herts.
1874. *Hall, Francis de Havilland, M.D., 47, Wimpole-street, W. VP 2, LL, C 7, S 2, SM.
1891. Handfield-Jones, Montagu, M.D., 35, Cavendish-square, W.
1891. Harold, John Patrick, M.R.C.S., 91, Harley-street, W.

1882. Harper, Gerald Samuel, M.B., 40, Curzon-street, Mayfair, W. c.
1895. Harrison, Charles James, M.D., Rossetti-mansions, Chelsea, S.W.
1871. Harrison, Reginald, F.R.C.S., 6, Lower Berkeley-street, Portman-square. p, vp 2, ll, c 2.
1883. *Hartridge, Gustavus, F.R.C.S., 12, Wimpole-street, W.
1882. Harvey, John Stephenson Selwyn, M.D., 1, Astwood-road, Cromwell-road, S.W.
1882. Haslam, William Frederick, F.R.C.S., 54, Newhall-street, Birmingham. c 3.
1852. *Haward, Edwin, M.D., 34a, Gloucester-place, W.
1898. Haydon, Arthur George, M.D., 28, Wimpole-street, W.
1897. Hayman, Albert Stephen, L.R.C.P., 26, Welbeck-street, W.
1899. Hays, Alexander Glencorse, L.R.C.P., Eydon, Byfield, Northamptonshire.
1890. Hebb, Frederick Theodore, M.R.C.S., 98, Oakley-street, Chelsea-embankment, S.W.
1897. Hedley, William Snowdon, M.D., 8, Mansfield-street, W.
1891. Hensman, Frank, M.R.C.S., Lieut.-Colonel, R.A.M.C., Underhill, Sandgate, Kent.
1883. Herman, George Ernest, M.B., 20, Harley-street, W.
1879. Heron, George Allan, M.D., 57, Harley-street, Cavendish-square, W. c 3.
1883. Herschell, George A., M.D., 76, Wimpole-street, W.
1883. Hewitt, Frederick William, M.D., 14, Queen Anne-street, W. c.
1892. Hill, William, M.D., 28, Weymouth-street, W.
1873. Hobson, William Henry, M.R.C.S., 38, Leinster-gardens, Hyde Park, W.
1895. Hodgson, George G. Graham, M.R.C.S., The Cedars, Chertsey, Surrey.
1879. Hogg, Arthur John, M.R.C.S., Leslie Lodge, Haven-green, Ealing, W.
1884. Holland, Charles Edward, M.B., "Airdrie," The Avenue, Kew Gardens, Surrey.
1868. Holman, Constantine, M.D., 26, Gloucester-place, Portman-square, W. vp 2, c 5.
1897. Holroyde, John, M.R.C.S., Camden House, Chatham.
1881. Hood, Donald William Charles, M.D., 43, Green-street, W. c 2.
1896. Horsley, Victor, F.R.C.S., F.R.S., 25, Cavendish-square, W. fm.
1883. *Hovell, T. Mark, F.R.C.S. Edin., 105, Harley-street, W.
1886. Huddart, Cuthbert Henry Cooke, M.B., Shoyswell Manor, Etchingham, Sussex.
1890. Hughes, Edgar, F.R.C.S., 91, Onslow-gardens, South Kensington, S.W.
1864. Hume, Frederick Henry, M.D., 53, Devonshire-street, Islington, N.
1884. Hunter, Sir Guyer, M.D., K.C.M.G., East India U.S. Club, St. James's, S.W.
1889. Hunter, William, M.D., 103, Harley-street, W.

1881. Hutchinson, Jonathan, F.R.C.S., F.R.S., 15, Cavendish-square. P, LL, C 5, O.
1892. Hutchinson, Jonathan, Jun., F.R.C.S., 1, Park-crescent, Portland-place, W.
1875. Hutchinson, Samuel John, M.R.C.S., 64, Brook-street, W.
1897. Hutchison, Robert, M.D., Toynbee Hall, Commercial-road, E.
1895. Hyde, Samuel, M.D., Lismore House, Buxton, Derbyshire.

1889. I'Anson, William Andrew, M.R.C.S., Denton Hall, Newcastle-on-Tyne.
1891. Isaac, George Washington, M.B., 75, Gower-street, W.C. *Councillor.*

1884. *Jackson, Frederick William, M.D., Yorkgate House, Broadstairs.
1868. Jackson, John Hughlings, M.D., F.R.S., 3, Manchester-square, W. P, VP, O, C 5.
1899. Jacobs, Henry Ralph, M.R.C.S., Culworth, Banbury, Oxon.
1898. Jaffrey, Francis, F.R.C.S., 21, Bentinck-street, Cavendish-square, W.
1887. Jamison, Arthur Andrew, M.D., 18, Lowndes-street, Belgrave-square, S.W.
1884. Jennings, Charles Egerton, M.D., 48, Seymour-street, W.
1886. Jervis, Arthur, M.D., 52, York-street, Portman-square, W.
1883. Jessop, Walter Hamilton, F.R.C.S., 73, Harley-street, W.
1893. Johnston, George Francis, M.D., 11, Upper Berkeley-street, W.
1886. Johnston, James, M.D., 53, Prince's-square, Bayswater, W.
1893. Joll, Boyd Burnett, M.B., 27, Bedford-square, W.C.
1888. Jones, Arthur Henry, M.D., 45, Sheep-street, Northampton.
1899. Jones, E. J. Trevor, M.D., Ty-mawr, Aberdare, S. Wales.
1893. Jones, Henry Lewis, M.D., 61, Wimpole-street, W.
1890. Jones, H. Macnaughton, M.D., 131, Harley-street, Cavendish-square, W.
1888. Jones, John Talfourd, M.B., Rosebank, South-terrace, Eastbourne, Sussex.
1892. *Jones, Robert, F.R.C.S., 11, Nelson-street, Liverpool.
1896. Jones, Robert, M.D., Claybury Asylum, Woodford Bridge, Essex.
1893. Joule, John Samuel, M.D., 32, Maida-hill West, W.
1877. Juler, Henry Edward, F.R.C.S., 23, Cavendish-square, W. C 2.

1889. Kauffmann, Otto Jackson, M.D., 22, Broad-street, Birmingham.
1891. Keegan, Denis Francis, M.D., Brigade Surgeon-Lieutenant-Colonel, East India United Service Club, St. James's-square, S.W.
1896. Keep, A. Corrie, M.C., 14, Gloucester-place, Portman-square, W.
1884. Keetley, Charles Bell, F.R.C.S., 56, Grosvenor-street, W. C 3.
1899. Kellock, Thomas Herbert, F.R.C.S., 8, Queen Anne-street, W.
1847. *Kellock, William Berry, M.D., 94, Stamford-hill, N.
1891. Kelson, William Henry, M.D., 96, Queen-street, Cheapside, E.C.
1890. Ker, Hugh Richard, F.R.C.S. Edin., Tintern, 2, Balham-hill, S.W.
1886. Kidd, Percy, M.D., 60, Brook-street, W. C 2.

1895. King, Arthur, M.B., Belmont, Station-road, Watford, Herts.
1897. Kingscote, Ernest, M.B., 31, Lower Seymour-street, W.
1883. Knapton, George, L.R.C.P. Edin., Craven House, Moss-lane, Manchester.

1898. Lack, H. Lambert, M.D., 48, Harley-street, W.
1889. Lake, Richard, F.R.C.S., 19, Harley-street, W.
1895. Lane, James Ernest, F.R.C.S., 46, Queen Anne-street, W.
1881. Langton, John, F.R.C.S., 62, Harley-street, W. c 2.
1890. Law, Edward, M.D. Edin., 35, Harley-street, W.
1858. *Lawson, George, F.R.C.S., 12, Harley-street, W. vp 2, c 3.
1893. Lee, E. Samuel, M.D., 31, Pevensey-road, St. Leonards-on-Sea.
1887. Leggatt, Charles Ashley Scott, M.D., 2, Walton-place, S.W.
1894. Lendon, Edwin Harding, M.B., 162, Holland Park-avenue, W.
1886. Lewers, Arthur Hamilton Nicholson, M.D., 72, Harley-street, W.
1895. Lewis, Ernest Wool, M.R.C.S., 12, King-street, Hammersmith, W.
1867. Lichtenberg, George, M.D., 47, Finsbury-square, E.C. c 2.
1898. Ling, Maurice Edward, M.R.C.S., 5, West Halkin-street, S.W.
1878. Lister, the Right Hon. Lord, D.C.L., LL.D., F.R.C.S., President of the Royal Society, 12, Park-crescent, Portland-place, W. o.
1896. Lister, Thomas D., F.R.C.S., 95, Wimpole-street, W.
1889. Little, Ernest Muirhead, F.R.C.S., 40, Seymour-street, Portman-square, W.
1889. Little, Fletcher, M.D., 32, Harley-street, W.
1878. Lockwood, Charles Barrett, F.R.C.S., 19, Upper Berkeley-street, W. vp 2, c 2, s 2.
1873. Loe, James Scarborough, M.R.C.S., Grange-avenue, Chapeltown-road, Leeds.
1881. Lorimer, G., M.D., 9, Terrace-road, Buxton, Derbyshire.
1899. Low, Richard Marsden Pilkington, M.B., 70, Philbeach-gardens, S.W.
1899. Low, Vincent Warren, F.R.C.S., 27, Queen Anne-street, W.
1868. Lowe, John, M.D., J.P., 4, Gloucester-place, Portman-square, W. c 3.
1897. Luff, Arthur Pearson, M.D., 31, Weymouth-street, W.
1889. Lunn, Henry Simpson, M.D., 5, Endsleigh-gardens, N.W.
1889. Lush, Percy, M.B., 4, Maresfield-gardens, Hampstead, N.W.
1899. Lynes, John, M.R.C.S., 2, Argyll-road, Kensington, W.

1884. MacBryan, Henry Crawford, L.R.C.P. Edin., Kingsdown House, Box, Wilts.
1894. McCann, Frederick John, M.B., 5, Curzon-street, Mayfair, W.
1885. McConnel, Henry Wilson, M.B., Great Ryburgh, Fakenham, Norfolk.
1871. MacCormac, Sir William, Bart., K.C.V.O., 13, Harley-street, W., President of the Royal College of Surgeons. p, vp, § 2, c 4, o.

1885. MacGeagh, Thomas Edwin Foster, M.D., 23, New Cavendish-street, W.
1896. Macgregor, Patrick Fraser, M.D., "Ashley," Cambridge Park, Wanstead, Essex.
1873. McHardy, Malcolm Macdonald, F.R.C.S. Edin., 5, Savile-row, W.
1894. Mackenzie, Hector William Gavin, M.D., 59, Welbeck-street, W. c.
1880. Mackenzie, Stephen, M.D., 18, Cavendish-square, W. vp 2, c 4, ll.
1881. Maclagan, Thomas John, M.D., 9, Cadogan-place, S.W. c 3.
1861. MacLaren, Alexander Connell, 60, Harley-street, W.
1891. Maclean, Allan, 10, Mitre-court Chambers, Temple, E.C.
1887. Macready, Jonathan Foster Christian Horace, F.R.C.S., 42, Devonshire-street, W. c 2.
1883. Maddick, Edmund Distin, F.R.C.S. Edin., 5, Cavendish-square, W.
1885. Maguire, Robert, M.D., 4, Seymour-street, W. c 3, s 2. *Councillor.*
1898. Mahomed, Arthur George Sulieman, M.R.C.S., Astolat, Poole-road, Bournemouth.
1890. Malcolm, John David, F.R.C.S. Edin., 13, Portman-street, Portman-square, W.
1893. Malcolm, William A., M.B., Oak House, 421, Holloway-road, N.
1887. Mantle, Alfred, M.D., Savile-place, Halifax.
1888. Mapother, Edward Dillon, M.D., 32, Cavendish-square, W. c 3.
1898. Marriott, Charles William, M.D., Aubrey House, Bath-road, Reading.
1891. Marsh, Howard, F.R.C.S., 30, Bruton-street, W.
1869. Marshall, William, M.D., Torrieburn, Barnes, S.W. c.
1889. Martin, John Michael Harding, M.D., Arnheim, Blackburn, Lancashire.
1890. Martin, Sidney, M.D., F.R.S., 10, Mansfield-street, W. c. *Hon. Librarian.*
1893. Mason, George Armstrong, M.B., 45, George-street, Portman-square, W.
1884. Matheson, Farquhar, M.B., 11, Soho-square, W.
1891. Maude, Arthur, M.R.C.S., Winterton House, Westerham, Kent.
1892. May, Chichester Gould, M.D., 59, Cadogan-place, S.W.
1891. May, William Page, M.D., 49, Welbeck-street, W.
1884. Meredith, William Appleton, F.R.C.S., 21, Manchester-square, W. c.
1897. Miles, William Ernest, F.R.C.S., 80, Ladbroke-grove, Notting-hill, W.
1895. Monier-Williams, Montagu Sneade Faithfull, M.R.C.S., 54, Onslow-gardens, S.W.
1883. Moore, Thomas, F.R.C.S., 6, Lee-terrace, Blackheath, S.E.
1883. Morgan, John Hammond, F.R.C.S., 68, Grosvenor-street, W. vp 2, ll, s 2, c. *Councillor.*
1899. Morier, Charles G. D., L.R.C.P.I., 1, Hamilton-terrace, N.W.
1893. Morison, Alexander, M.D., 14, Upper Berkeley-street, W.

1871. Morley, Alexander, 42, Albemarle-street, W.
1895. Morley, Frank, M.R.C.S., 42, Albemarle-street, W.
1881. Morris, Henry, F.R.C.S., 8, Cavendish-square, W. c.
1878. Morris, Malcolm Alexander, F.R.C.S. Edin., 8, Harley-street, W. c 2.
1882. Morton, Andrew Stanford, F.R.C.S., 133, Harley-street, W.
1884. *Moullin, Charles William Mansell, F.R.C.S., 69, Wimpole-street, W.
1899. Mullick, Sarat Kumar, M.B., 52, Margaret Street, W.
1894. Murray, Charles Stormont, L.R.C.S. Edin., 85, Gloucester-place, W.
1884. Murray, Fred., M.B., Durbanville, Cape Colony, South Africa.
1890. Murray, George, M.R.C.S., 34, Wimpole-street, Cavendish-square, W.
1886. Murray, Hubert Montague, M.D., 25, Manchester-square, W. c 3.
1879. Murrell, William, M.D., 17, Welbeck-street, W.

1894. Naumann, J. C. Francis, M.D., 125, Gower-street, W.C.
1889. *Nias, J. Baldwin, M.D., 5, Rosary-gardens, South Kensington, S.W. c. *Councillor.*
1880. Nix, Edward James, M.D., 11, Weymouth-street, W. c 3.

1887. Oakley, Adam Robert Hamilton, L.R.C.P., Treath, Hornchurch, Essex.
1898. O'Brien, Christopher, M.D., M.R.C.S., 135, North Strand-road, Dublin.
1894. O'Callaghan, Robert, F.R.C.S.I., 137, Harley-street, W.
1885. Ogilvie, Leslie, M.B., 46, Welbeck-street, W.
1884. Ogle, Charles John, M.R.C.S., 1, Cavendish-place, W.
1884. Oliver, George, M.D., West End Park, Harrogate.
1892. Openshaw, Thomas Horrocks, F.R.C.S., 16, Wimpole-street, W.
1875. Ord, William Miller, M.D., 37, Upper Brook-street, W. p, o, c 4.
1892. Ord, William Wallis, M.D., The Hall, Salisbury.
1887. Ormerod, Joseph Arderne, M.D., 25, Upper Wimpole-street, W. c.
1889. Orton, George Hunt, M.B., 1a, Campden Hill-road, Kensington, W.
1884. Orwin, Arthur Wigelsworth, M.D., 15, Weymouth-street, Portland-place, W.
1894. Osborn, Samuel, F.R.C.S., J.P., 1a, Devonshire-street, Portland-place, W.
1880. Oswald, James Waddell Jeffreys, M.D., 245, Kennington-road, S.E. c 3.
1883. Owen, Charles J. Rayley, M.R.C.S., 14, Devonshire-terrace, Hyde Park, W. c.
1878. *Owen, Edmund, F.R.C.S., 64, Great Cumberland-place, W. p, o, vp 2, c 3, s 2, sm, ll. *Trustee.*
1881. Owen, Isambard, M.D., 40, Curzon-street, Mayfair, W. s 2, c 4.

1880. Palmer, Frederick Stephen, M.D., Compton Lodge, East Sheen, S.W.
1882. Palmer, William Pitt, M.B., Kirkham, Babbacombe, Torquay.
1877. *Paramore, Richard, M.D., 2, Gordon-square, W.C.
1881. Parrott, Edward John, M.R.C.S., The Thorn, Hayes, Middlesex.
1871. Parsons, Francis Henry, M.D., "The Hurst," West Worthing.
1885. Pasteur, William, M.D., 4, Chandos-street, Cavendish-square, W. s 2, c. 2. *Trustee.*
1872. Patten, Charles Arthur, M.R.C.S., Marpool House, Ealing, W.
1891. Patterson, Charles Sumner, M.B., 40, Highbury-place, N.
1890. Pattison, Edward Seton, M.R.C.S., Granville House, Fulham-road, S.W.
1854. *Pavy, Frederick William, M.D., F.R.S., 35, Grosvenor-street, W. vp, ll, c.
1881. *Peacey, William, M.D., Rydal Mount, St. John's-road, Eastbourne.
1883. Peck, Edward George, M.A., The Willows, Queensbury, Bradford, Yorks.
1871. Pedler, George Henry, M.R.C.S., 6, Trevor-terrace, Knightsbridge, S.W.
1883. Perigal, Arthur, M.D., New Barnet, Herts.
1878. Philipps, Sutherland Rees, M.D., St. Ann's Heath, Virginia Water, Berks.
1894. Phillipps, William Alfred, M.D., 13, John-street, Berkeley-square, W.
1876. Phillips, Charles Douglas Ferguson, M.D., F.R.S.E., 10, Henrietta-street, Cavendish-square, W. c 3.
1873. Phillips, George Richard Turner, M.R.C.S., 24, Palace-court, Bayswater-hill, W. c 2.
1883. Phillips, Sidney Philip, M.D., 62, Upper Berkeley-street, Portman-square, W.
1883. Pick, Thomas Pickering, F.R.C.S., 18, Portman-street, W. c 2.
1883. Pitts, Bernard, F.R.C.S., 109, Harley-street, Cavendish-square. c 5, s 2.
1890. Pope, Harry Campbell, M.D., 280, Goldhawk-road, Shepherd's Bush, W.
1871. Powell, Sir Richard Douglas, Bart., M.D., 62, Wimpole-street, W. p, vp, c 5, o.
1891. Powell, William Wyndham, F.R.C.S., 16, Old Burlington-street, W.
1891. Preston, Theodore Julian, M.R.C.S., Fleet Surgeon, Royal Navy, 238, Portsdown-road, Maida Vale, W.
1891. *Prickett, Marmaduke, M.D., 27, Oxford-square, W.
1885. Pringle, John James, M.B., 23, Lower Seymour-street, W.
1889. Pritchard, Owen, M.D., 41, Gloucester-square, W.
1898. Probyn-Williams, Robert James, M.D., 13, Welbeck-street, W.

1892. Ramsay, James, M.D., High Peter-gate, York.
1894. Rankin, Guthrie, M.D., 4, Chesham-street, Belgravia, S.W.

1881. Ranking, John Ebenezer, M.D., Hanover House, Tunbridge Wells.
1894. Rayner, Herbert Edward, F.R.C.S., Harcourt House, Camberley, Surrey.
1859. *Rayner, John, M.D., Swaledale House, Highbury-quadrant, N.
1890. Reid, John, M.B., Clanmurray, Dromore, co. Down.
1882. Reid, Thomas Whitehead, M.D., 34, St. George's-place, Canterbury.
1872. *Richards, Joseph Peeke, M.R.C.S., 6, Freeland-road, Ealing, W. c 3.
1899. Richardson, Sidney William F., F.R.C.S., St. Thomas's Hospital, S.E.
1868. *Roberts, David Lloyd, M.D., F.R.S.E., 11, St. John's-street, Manchester.
1857. *Roberts, David Watkin, M.D., 56, Manchester-street, W.
1885. Roberts, Edward Coleridge, M.R.C.S., Southgate, N.
1874. Roberts, Frederick Thomas, M.D., 102, Harley-street, W. vp 2, o, ll, c. *President.*
1884. Robinson, Arthur Henry, M.D., Mile End Infirmary, Bancroft-road, E.
1897. Robinson, Henry Betham, M.S., 1, Upper Wimpole-street, W.
1896. Robson, A. Mayo, F.R.C.S., 7, Park-square, Leeds.
1847. *Rogers, William Richard, M.D., 26, Upper Baker-street, N.W. vp, c 6.
1897. Rolleston, Humphry Davy, M.D., 112, Harley-street, W. c. *Councillor.*
1890. Root, Arthur Guernsey, M.D., 46, Eagle-street, Albany, New York, U.S.A.
1886. Rose, Robert Duncan, F.R.C.S., 8, St. Leonards, York.
1874. Rose, William, F.R.C.S., 17, Harley-street. c 2, ll, vp 2.
1883. *Ross, Daniel McClure, M.D., Cedar Lodge, Littledown-road, Bournemouth.
1888. *Roth, Bernard, F.R.C.S., J.P., 38, Harley-street, W.
1893. Roughton, Walter, F.R.C.S., Cranborne House, New Barnet, Herts.
1881. *Routh, Amand, M.D., 14a, Manchester-square, W. c 5, s 2.
1848. *Routh, Charles Henry Felix, M.D., 52, Montagu-square, W. p, vp 2, o, ll, s 4, c 6, sm. *Trustee.*
1891. Ruffer, Marc Armand, M.D., Medical School, Cairo, Egypt.
1887. Rushworth, Frank, M.D., "Langdale," Goldhurst-terrace, South Hampstead, N.W.
1889. Russell, James Samuel Risien, M.D., 4, Queen Anne-street, W.
1886. Rutherfoord, Henry Trotter, M.D., Salisbury House, Taunton.

1887. Sainsbury, Harrington, M.D., 63, Welbeck-street, W.
1863. *Sansom, Arthur Ernest, M.D., 84, Harley-street, W. p, vp, s 2, c 6, sm, §, ll, o.

1895. Sansom, Charles Lane, F.R.C.S. Edin., 5, Grosvenor-gardens, Grosvenor-place, S.W.
1897. Sansom, Harry Arthur, M.D., The Glen, West End-lane, West Hampstead, N.W.
1886. Savage, George Henry, M.D., 3, Henrietta-street, W. c.
1886. Savill, Thomas Dixon, M.D., 60, Upper Berkeley-street, W.
1899. Sayres, Alexander Ward Fortescue, M.D., Woodford, N.E.
1899. Schorstein, Gustave, M.B., 11, Portland-place, W.
1873. Sedgwick, James, M.D., Boroughbridge, Yorkshire.
1868. Sedgwick, Leonard William, M.D., 48, Gloucester-terrace Hyde Park, W. vp 2, c 4, § 3.
1883. Semon, Sir Felix, M.D., 39, Wimpole-street, W. §, c.
1887. Servais, Leopold, M.D., Antwerp, Belgium.
1889. Shaw, George, M.B., 1, The Drive, West Brighton.
1884. Shaw, John, M.D., 32, New Cavendish-street, Cavendish-square, W.
1886. Sheild, Arthur Marmaduke, F.R.C.S., 4, Cavendish-place, W. c 2, s 2.
1890. Sheppard, William John, M.D., 211, Upper Richmond-road, Putney, S.W.
1881. Shipton, Arthur, F.R.C.S. Edin., The Square, Buxton, Derbyshire.
1885. Shoemaker, John V., M.D., 1031, Walnut-street, Philadelphia, U.S.A.
1894. Shuttleworth, George Edward, M.D., Ancaster House, Richmond, Surrey.
1890. Silk, John Frederick William, M.D., 47, Devonshire-street, W.
1884. Simpson, James Herbert, M.D., Hillmorton-road, Rugby, Warwickshire.
1898. Simpson, John Pollock, M.D., 1, Blandford-street, Manchester-square, W.
1884. Sinclair, John, M.R.C.P., General Post Office, St. Martin's-le-Grand, E.C.
1891. Sisley, Richard, M.D., 14, Park-lane, W.
1883. *Skerritt, Edward Markham, M.D., Richmond Hill, Clifton.
1886. Slater, Charles, M.B., 81, St. Ermin's-mansions, Westminster, S.W.
1862. Slight, George, M.D., 14, Old Burlington-street, W. c 2.
1889. Smale, Morton, M.R.C.S., 22A, Cavendish-square, W. c 2. *Councillor.*
1845. *Smiles, William, M.D., St. Martha's Lodge, Guildford. vp 2, s 4, c 9, sm.
1897. Smith, A. Lionel H., M.R.C.S., Addenbrooke's Hospital, Cambridge.
1896. Smith, E. Stanley, M.D., 68, Wimpole-street, W.
1887. Smith, Frederick John, M.D., 138, Harley-street, W. *Councillor.*
1873. Smith, Heywood, M.D., 18, Harley-street, W. c 3.
1880. Smith, Noble, F.R.C.S. Edin., 24, Queen Anne-street, W.
1891. Smith, Solomon Charles, M.D., Four Oaks, Walton-on-Thames.
1877. Smith, Sydney Lloyd, M.R.C.S., 25, Argyle-square, King's Cross, W.C.
1882. Smith, Thomas Frederick Hugh, F.R.C.S., Farningham, Kent.

1873. *Smith, Thomas Gilbart, M.D., 68, Harley-street, W. vp 2, s 2, sm, c 4. *Trustee.*

1872. Smith, Walter, M.R.C.P. Edin., Orleans House, 60, Regent's Park-road, N.W.

1874. Smyth, William Woods, L.R.C.P. Edin., Maidstone.

1894. Snape, Ernest Alfred, M.D., 41, Welbeck-street, W.

1893. Spencer, Herbert Ritchie, M.D., 104, Harley-street, W. *Councillor.*

1887. Spicer, Scanes, M.D., 28, Welbeck-street, W.

1883. Spitta, Edmund Johnson, M.R.C.S., Ivy House, Clapham Common, S.W.

1864. Squire, Alexander John Balmanno, M.B., 24, Weymouth-street, Portland-place.

1881. Startin, James, M.R.C.S., 15, Harley-street, W.

1892. Staveley, William H. C., F.R.C.S., 13, South Eaton-place, S.W.

1896. Steele-Perkins, George Chapman, M.B., 85, Wimpole-street, W.

1884. Stephens, William John, L.S.A., 9, Old Steine, Brighton.

1882. Stewart, James, F.R.C.P. Edin., Dunmurry, Sneyd Park, near Clifton.

1894. Stewart, Kenneth Trevor, M.D., 12, Chapel-street, Belgrave-square, S.W.

1883. Stewart, William Robert Henry, F.R.C.S. Edin., 42, Devonshire-street, Portland-place, W.

1884. Stiven, Edward Winnan Fleming, M.D., Lincoln House, Harrow, Middlesex.

1885. Stivens, B. H. Lyne, M.D., 107, Park-street, Grosvenor-square, W.

1848. *Stocker, John Sherwood, M.D., 2, Montagu-square, W. c 10, s 2.

1884. Stoker, George, M.R.C.S., J.P., 14, Hertford-street, Mayfair, W.

1892. Stonham, Charles, F.R.C.S., 4, Harley-street, W.

1877. Stowers, James Herbert, M.D., 128, Harley-street, W.

1873. Strange, William Heath, M.D., 2, Belsize-avenue, Hampstead, N.W. c 3.

1881. Sturge, William Allen, M.D., Maison Malausséna, Boulevard Dubouchage 29, Nice. sm.

1889. Sumpter, Walter John Ernely, M.R.C.S., Sheringham, Norfolk.

1892. Sunderland, Septimus, M.D., 11, Cavendish-place, W.

1876. *Sutherland, Henry, M.D., 21, New Cavendish-street, W.

1892. Sutton, J. Bland, F.R.C.S., 48, Queen Anne-street, W.

1896. Swanton, James Hutchinson, M.D., Causey Ware Hall, Lower Edmonton, N.

1892. Swift, William John Cropley, M.R.C.S., 4, Gordon-square, W.C.

1885. *Syers, Henry Walter, M.D., 75, Wimpole-street, W.

1884. Symonds, Horatio Percy, F.R.C.S., 35, Beaumont-street, Oxford.

1893. Symonds, Charters James, F.R.C.S., 58, Portland-place, W. *Councillor.*

1899. *Sympson, Edward Mansel, M.D., Deloraine Court, Lincoln.

1864. Tait, Edward Wilmshurst, M.R.C.S., 48, Highbury-park, N.
1875. Tamplin, Charles Harris, M.R.C.S., 17, Paragon, Ramsgate.
1882. Taylor, Seymour, M.D., 16, Seymour-street, Portman-square, W. c 2.
1897. Templeton, George, F.R.C.S., 8, Mansfield-street, W.
1898. Thomas, John Lynn, F.R.C.S., 28, Charles-street, Cardiff.
1859. *Thompson, Edmund Symes, M.D., 33, Cavendish-square, W. vp, o, s 3, c 3, sm.
1855. *Thompson, Sir Henry, Bart., F.R.C.S., 35, Wimpole-street, W. vp, ll, c 4.
1896. Thompson, Henry George, M.D., 86, Lower Addiscombe-road, Croydon, Surrey.
1895. Thompson, James Andrew Baird, M.D., White Hall, Abridge, Essex.
1873. Thomson, John Roberts, M.D., Monkchester, Bournemouth, Hants.
1894. Thomson, St. Clair, M.D., 28, Queen Anne-street, W.
1898. Thorne, Atwood, M.B., 10, Nottingham-place, W.
1897. Thornton, Bertram, M.R.C.S., J.P., Berkeley Lodge, Trinity-square, Margate.
1892. Thornton, George, M.D., Fountain Hospital, Lower Tooting, S.W.
1876. Thornton, John Knowsley, M.C., J.P., Hildersham Hall, Cambs. p, vp, c 3.
1867. Thorowgood, John Charles, M.D., 61, Welbeck-street, W. ll, s 2, sm, c 5, vp 4.
1856. *Thudichum, John Louis William, M.D., 11, Pembroke-gardens, Kensington, W. vp, ll, o, c.
1884. Thursfield, Thomas William, M.D., J.P., Selwood, Beauchamp-square, Leamington.
1865. Travers, William, M.D., 2, Phillimore-gardens, Kensington, W.
1897. Tresilian, Frederick James, M.D., Redlingtons, Enfield.
1884. *Treves, Frederick, F.R.C.S., 6, Wimpole-street, W. c 3, ll, vp 2.
1899. Tubby, Alfred Herbert, M.S., 25, Weymouth-street, W.
1898. Tucker, E. F. Gordon, M.R.C.S., Indian Medical Service, care of Messrs. H. King and Co., 45, Pall Mall, S.W.
1882. Tuke, Charles Molesworth, M.R.C.S., Chiswick House, Chiswick.
1886. Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.
1884. Turner, George R., F.R.C.S., 41, Halfmoon-street, Piccadilly, W. s 2, c 3. *Vice-President.*
1890. Tweed, Edward Reginald, M.D., Hembury Fort, Honiton, Devon.
1883. Tweedy, John, F.R.C.S., 100, Harley-street, W. c.
1891. Tyson, William Joseph, M.D., 10, Langhorne-gardens, Folkestone.

1887. *Underwood, Edward T., M.D., Fort Bombay, India.

1897. Vassie, Alexander Henry, M.B., 98, Priory-road, West Hampstead, N.W.
1883. Venning, Edgcombe, F.R.C.S., 30, Cadogan-place, S.W.

1899. Vise, Christopher, M.D., 39, Mount Pleasant-road, Tunbridge Wells.
1893. Voelcker, Arthur Francis, M.D., 31, Harley-street, W. *Councillor.*

1892. Waggett, Ernest Blechynden, M.B., 45, Upper Brook-street, W.
1850. *Waggett, John, M.D., Perivale, Bournemouth.
1884. Wakley, Thomas, Jun., L.R.C.P. Lond., 5, Queen's-gate, S.W.
1850. *Wakley, Thomas Henry, F.R.C.S., 5, Queen's-gate, S.W.
1898. Wallace, Cuthbert Sidney, F.R.C.S., St. Thomas's Hospital, S.E.
1894. Wallis, Frederick Charles, F.R.C.S., 26, Welbeck-street, W. c. *Hon. Secretary.*
1880. Walsham, William Johnson, F.R.C.S., 77, Harley-street, W. c.
1895. Walters, Frederick R., M.D., 21, Wimpole-street, W.
1899. Ward-Humphreys, George Herbert, M.R.C.S., 16, Cavendish-square, W.
1894. Waring, Holburt Jacob, F.R.C.S., 9, Upper Wimpole-street, W.
1899. Washbourn, John Wickenford, M.D., 6, Cavendish-place, W.
1894. Waterhouse, Herbert Furnivall, F.R.C.S., 81, Wimpole-street, W.
1883. Waterhouse, William Dakin, LL.D., 50, West End-lane, West Hampstead, N.W. c.
1891. Watson, W. Spencer, F.R.C.S., 1, Duke-street, Manchester-square, W.
1889. Waugh, Henry Dunn, M.D., 6, Sumner-place, Onslow-square, S.W.
1884. Webb, F. Ernest, M.R.C.S., 113, Maida-vale, W.
1896. Weber, Frederick Parkes, M.D., 19, Harley-street, W.
1889. Weber, Sir Hermann, M.D., 10, Grosvenor-street, W.
1898. Weeks, Courtenay Charles, M.R.C.S., Pinchbeck, Spalding, Lincs.
1838. *Wells, John Robinson, F.R.C.S., 4, Pierrepoint-road, Acton, W. c 2.
1884. West, Samuel, M.D., 15, Wimpole-street, W. s 2, c 4, cfc, ll. *Vice-President.*
1889. Wethered, Frank Joseph, M.D., 83, Harley-street, W.
1892. Wheaton, Samuel Walton, M.D., 76, The Chase, Clapham Common, S.W.
1882. Whipham, Thomas T., M.D., 11, Grosvenor-street, W. sm, c.
1884. Whistler, William MacNeill, M.D., 18, Wimpole-street, W.
1899. White, Charles Percival, M.B., 144, Sloane-street, S.W.
1889. White, E. F., F.R.C.S., Westlands, 280, Upper Richmond-road, Putney, S.W.
1880. *White, William Henry, M.D., 43, Weymouth-street, W. c. 3.
1883. Whitehead, Walter, F.R.C.S. Edin., F.R.S.E., 499, Oxford-road, Manchester. c.
1885. Whitla, William, M.D., 8, College-square North, Belfast, Ireland.
1877. Whitmore, William Tickle, F.R.C.S. Edin., 7, Arlington-street, Piccadilly, W.
1898. Willey, F. Ingor, M.B., The Wych, 8, Avenue-road, Highgate, N.
1872. Williams, Charles Theodore, M.D., 2, Upper Brook-street, Grosvenor-square, W. p, vp 2, ll, s 2, sm, o, l 3, c 9.

1876. Williams, Henry William, M.D., Hillside, Guilsborough, Northampton.
1883. Williams, Sir John, Bart., M.D., 63, Brook-street, W. c 3.
1873. Wills, Thomas Munns, F.R.C.S.I., J.P., 44, Merton-road, Bootle, Liverpool.
1893. Wills, William Alfred, M.D., 29, Lower Seymour-street, W.
1892. Wilson, Claude, M.D., Belmont, Tunbridge Wells.
1884. Winslow, H. Forbes, M.D., 14, York-place, Portman-square, W.
1873. Winslow, Lyttleton Stewart Forbes, M.B., D.C.L., LL.D., 33, Devonshire-street, W. c.
1876. Woakes, Edward, M.D., 78, Harley-street, W.
1886. Wood, T. Outterson, M.D., 40, Margaret-street, Cavendish-square, W. c 3.
1873. Woodhouse, Robert Hall, M.R.C.S., 1, Hanover-square, W.
1891. Woollett, Charles Jerome, F.R.C.S., "Ambleside," Streatham, S.W.
1898. Wright, Sydney Faulconer, M.D., St. Peter's Lodge, Eltham-road, Lee, S.E.
1884. Wyman, William Sanderson, M.D., Red Brae, Putney-hill, S.W. c 2.

1891. Yarr, Michael Thomas, Major, R.A.M.C., 91, Victoria-street, S.W.
1884. Yeo, I. Burney, M.D., 44, Hertford-street, Mayfair, W.
1897. Young, Adam, M.R.C.S., 2, College-crescent, South Hampstead, N.W.
1897. Young, Williams H. Frome, M.R.C.S., Malcolm Corner, Worple-road, Wimbledon.
1884. Younger, Edward George, M.D., 19, Mecklenburgh-square, W.C. c. *Councillor.*

NON-SUBSCRIBING FELLOWS.

1868. Bateman, Sir Frederic, M.D., J.P., Upper-street, Giles-street, Norwich.
1872. Bell, John Hougham, M.D., Ventnor, Isle of Wight.
1868. Buckle, Fleetwood, M.D., Staff Surgeon, R.N., Merton Lodge, Southsea.
1870. Clouston, Thomas Smith, M.D., Royal Asylum, Morningside, Edinburgh. FM 1870.
1868. Folker, William Henry, F.R.C.S., Hanley, Staffordshire.
1869. Foster, Sir Walter B., M.D., M.P., 14, Temple-row, Birmingham.
1868. Fox, Charles Henry, M.D., 35, Heriot-row, Edinburgh.
1868. Gaine, Charles, 30, Gay-street, Bath.
1871. Glynn, Thomas Robinson, M.D., 62, Rodney-street, Liverpool.
1868. Knaggs, Samuel, 2, Bradley-lane, Huddersfield.
1869. Lees, Charles Alexander, M.D., Dep. Inspector-General, R.N.
1869. Mathews, Robert, Bickley, Kent.
1871. Maurice, Oliver Calley, M.D., J.P., 75, London-street, Reading.
1868. McIntyre, John, M.D., LL.D., Odiham, Hants.
1868. Nevins, John Birkbeck, M.D., 32, Princes-avenue, Liverpool.
1871. Ogle, William, M.D., The Elms, Derby.
1869. Philipson, George Hare, D.C.L., M.D., J.P., 7, Eldon-square, Newcastle-on-Tyne.
1869. Price, William Preston, M.D., 1, Ethelbert-crescent, Margate.
1869. Prior, Charles Edward, M.D., 4, Goldington-road, Bedford.
1869. Roberts, Bransby, M.D., Ash Grove, Eastbourne.
1869. Stear, Henry, Saffron Walden, Essex.
1869. Taylor, Charles Bell, M.D., 9, Park-row, Nottingham.
1869. Walker, John Swift, M.D., Hanley, Staffs.
1868. Wiblin, John, F.R.C.S., Wimborne, Dorset.

*** As it is very desirable that the foregoing Lists should be kept as accurately as possible, Fellows are requested to send notice of any corrections that may be necessary to the Secretaries or to the Registrar.

GENERAL MEETING.

May 8th, 1899.

EDMUND OWEN, F.R.C.S., *President*, in the Chair.

Report of the Council for the Session, 1898–99.

DURING the past year there has been little but the routine work of the Society demanding the attention of the Council.

The total number of Fellows is now 747, showing an increase of one as compared with last year. During the Session, 32 Ordinary and six Honorary Fellows have been elected. The names of the latter are:—Professor B. C. Brouardel and C. E. Potain, of Paris; Professors V. V. Pachoutin, C. A. Rauchfuss, and N. V. Sklifosoffsky, of St. Petersburg; and Professor W. Osler, of Baltimore, U.S.A.

There have been 12 resignations, and 10 Fellows have been struck off the roll for non-payment of subscriptions.

The Council regrets to report that 15 Fellows have died, including two Honorary Fellows, Sir William Jenner, Bart., and Dr. Charles J. Hare, a Past President and Trustee; seven Ordinary Fellows—Dr. John Brunton (a Past Vice-President), Mr. E. Cotterell, Dr. James Duncan (elected 1848), Dr. J. H. Paul, Dr. H. Pott, Sir William Roberts, F.R.S., and Dr. Herbert Smith, also six Corresponding and Non-Subscribing Fellows.

The Annual Oration, delivered by Dr. Frederick Roberts, was entitled "The Moscow Congress," and attracted a large audience.

Dr. Samuel West delivered an interesting and important series o Lettsomian Lectures on "Some of the Clinical Aspects of Granular Kidney."

The Fothergillian Gold Medal was awarded, on the recommendation of the Fothergillian Committee, to Dr. S. Monckton Copeman "in recognition of his researches on the preservative effects of glycerine upon vaccine lymph and of the benefits, in a practical sense, that have arisen therefrom."

The Anniversary Dinner, under the Chairmanship of the President was held as usual on March 8th, the birthday of John Fothergill. There was a record attendance, and the dinner was in every respect a great success.

During the year Dr. A. Ernest Sansom (President 1897–98) gave a donation of ten guineas to the funds of the Society.

Dr. W. Pasteur was appointed one of the Trustees of the Real Property in the place of Dr. Hare, deceased.

The Council regrets to report the resignation of the Honorary Librarian, Dr. W. H. Allchin, who has held the office for upwards of 17 years. The Society is indebted to him for much of the increased usefulness of the Library, and the Council wishes to record its hearty appreciation of his devoted services.

(Signed) EDMUND OWEN,
President.

Report of the House and Finance Committee for the Session, 1898–99.

The Committee have to report that the Society's premises are in a good state of repair, and all the rooms not occupied by the Society are let. The income from regular tenants has been £20 more than last year, but there has been a falling off in the fluctuating item of "occasional lettings."

Since the date of the last report two more debentures have been drawn and paid off, thus reducing the amount outstanding to £3,000.

The Committee note, with satisfaction, that there has been a considerable increase in the number of subscriptions paid by means of orders on the Fellows' bankers; and, in view of the saving of correspondence and labour which this mode of payment ensures, earnestly recommend its adoption by all the Fellows of the Society. Order forms for this purpose can be had on application to the Registrar.

(Signed) D. H. GOODSALL,
Chairman.

Honorary Librarian's Report, Session 1898–99.

I have the honour to report that the condition of the Library for the year ending April 30th, 1899, has been in all respects satisfactory. Its increasing usefulness to the Fellows of the Society is shown by the much larger number of volumes issued than has occurred in any previous year, 232 having been borrowed by Fellows and 59 volumes obtained from Lewis's Library. The success of the Society's action in subscribing to the latter is fully assured and, as was anticipated, has done much to develop the use of the Society's own Library.

There has been a considerable addition to the shelves during the past year. Of new works, 29 volumes have been presented by authors and publishers, and 17 purchased. In addition, 25 volumes of old works were presented by Dr. Gubb, four by the Hon. Librarian, and 12 were purchased, making a total of 87 volumes added to the Library.

Appended is a list of the principal works acquired,

A portion of the donation lately made to the funds of the Society by Dr. Robert Barnes having been assigned to Library uses, it was determined to expend a part at once in the cleaning and re-labelling of 270 of the older parchment-bound works. A sum of £36 still remains for disposal in a similar manner. By a grant from the Society's funds 12 of the old works have been rebound.

Some progress has been made in the card catalogue of the sixteenth and seventeenth century works.

In resigning the office of Honorary Librarian after 18 years' service, I would desire to express my acknowledgments to the Society for the readiness with which they have adopted the various suggestions I have made and the forbearance they have shown me in the performance of my duties. Much remains to be done in the arrangement and cataloguing of the older volumes, though a substantial start has been made, and it is with sincere regret that I have felt compelled, in the interests of the Library, to withdraw from work which I have not the leisure satisfactorily to carry out. I would again commend to the grateful consideration of the Society the admirable services of Mr. Hall, by whose painstaking and conscientious care the labours of my office have for many years been considerably lightened.

(Signed) W. H. Allchin,
Hon. Librarian.

Among the books added to the Library during the Session were the following :—

Allbutt (C.). 'System of Medicine.' Vol. vi.
Bennett (W. H.). 'Varix and Thrombosis.'
——— 'Appendicitis.'
Bryant (T.). 'Villous Growths.'
Cautley (E.). 'On Feeding Infants and Young Children.'
Gibson (G. A.). 'Diseases of the Heart and Aorta.'
Goodall (E. W.) and Washbourn (J. W.). 'Infectious Diseases.'
Goodhart (J. F.). 'Diseases of Children.' 6th edition.
Gowlland (P. Y.). 'Original Drawings Illustrating Rectal Surgery.'
Haig (A.). 'Diet and Food in Relation to Strength.'
Hall (F. de H.). 'Medical Examination for Life Insurance.'
Jaksch and Cagney (J.). 'Clinical Diagnosis.' 4th English edition.
Klein (E.). 'Elements of Histology.'
Lockwood (C. B.). 'Hernia, Hydrocele, and Varicocele.'
Luff (P.). 'Gout : Its Pathology and Treatment.'
Mapother (E. D.). 'Papers on Dermatology.' 2nd edition.
Morgan (J. H.). 'Affections of the Urinary Apparatus in Children.'
Morris (H.). 'Origin and Progress of Renal Surgery.'
——— 'Human Anatomy.' 2nd edition.
Morris (Malcolm). 'Diseases of the Skin.' 2nd edition.
Nathan (R.). 'The Plague in India, 1896–7.'
Powell (Sir R. Douglas). 'Treatment in Diseases of the Heart.'
Schafer (E. A.). 'Physiology.'
Sheild (A. M.). 'Diseases of the Breast.'
Smith (Greig). 'Abdominal Surgery.' 6th edition.

MEDICAL SOCIETY

DR. BALANCE SHEET, FROM 1ST OCTOBER,

RECEIPTS.

				£	s.	d.
Balance from Last Account				78	6	6
Ordinary—			£	s.	d.	
Subscriptions			508	14	6	
Entrance Fees			25	4	0	
Life Composition Fee			10	10	0	
Rents { Bolt Court (less expenses)	£80	16	6			
Rents { Chandos Street	717	3	0			
			797	19	6	
Occasional lettings			27	6	0	
Sale of 'Transactions'			2	12	6	
						1,372 6 6
Extraordinary—						
Donation from Dr. A. Ernest Sansom						10 10 0
						£1,461 3 0

FOTHERGILLIAN

	£	s.	d.
Balance from Last Account	63	15	11
Dividend on £916 10*s.* 5*d.*, $2\frac{3}{4}$ % Consols	25	4	0
	£88	19	11

(Signed) D. H. GOODSALL,
Treasurer.

October 2nd, 1899.

OF LONDON.

1898, to 30th September, 1899. Cr.

Expenditure.

Ordinary—				£	*s.*	*d.*	£	*s.*	*d.*
Rent, 11 and 12, Chandos Street				290	0	0			
Rates, Taxes, and Insurance				152	16	7			
Registrar's Salary and Allowances				117	13	6			
Collector's Poundage on Subscriptions				25	8	8			
Reporter's Salary				22	1	0			
Stationery and Printing				19	11	0			
Printing and issuing 'Transactions,' Vol. xxi ..				127	15	3			
Library Expenses				28	5	2			
Postage and Stamps				10	19	10			
Coals and Wood				9	7	10			
Gas and Electric Supply				43	12	11			
Repairs				22	3	7			
Interest on Debentures				118	11	8			
Conversazione				45	0	0			
Refreshments at Meetings				13	11	6			
Hire of Microscopes				1	8	0			
Bankers' Charges, 1*s.*, and Travelling, 4*s.* ..				0	5	0			
Wages				47	18	0			
Chandlery and Cleaning				13	14	5			
Anniversary Dinner	£103	15	6						
Less Fellows' Tickets	82	8	6						
				21	7	0			
							1,131	10	11
Extraordinary—									
Redemption of Two Debentures				200	0	0			
Gratuity to Registrar				25	0	0			
							225	0	0
Balance at Bank, 30th September, 1899				..		..	104	12	1
							£1,461	3	0

FUND.

	£	*s.*	*d.*
Gold Medal presented to Dr. Monckton Copeman..	21	0	0
Books Purchased for Library..	10	8	10
Balance at Bank, 30th September, 1899	57	11	1
	£88	19	11

Examined, compared with vouchers, and found correct,

(Signed) ROBERT MAGUIRE, } *Auditors.*
W. BRUCE CLARKE, }

October 4th, 1899.

TRANSACTIONS

OF THE

MEDICAL SOCIETY OF LONDON.

126TH SESSION.

October 10*th*, 1898.

REMARKS INTRODUCTORY TO THE WORK OF THE SESSION.

By the President, EDMUND OWEN, F.R.C.S.

GENTLEMEN,—It is customary for the President of the Medical Society of London, on his being installed in the Chair, first to offer thanks for the honour thus conferred upon him, and then to make some remarks in the shape of a little introductory address. To put myself in order, therefore, permit me to return my sincere and appreciative thanks for the honour which you have been so good as to confer upon me by electing me to this Chair which, 123 years ago, was so worthily and so ably filled by our Lettsom. And let me express the hope that during my year of office the Society may continue its career of prosperity and usefulness. Without any doubt the old Medical Society of London is prosperous: it is prosperous in its house and home, and in its long and influential list of Fellows; it is prosperous in the matter of its finance; and, above all, in regard to the feeling of good fellowship which permeates it root and branch. Certain of the Fellows are of opinion that the happy sense of fellowship which is indeed a marked feature of this Society is based upon the back of that historic turtle with which Lettsom feasted the young Society on an afternoon in June, 1775. This was one of the first of our annual dinners—be so kind as to remember that the 126th of them will be held at the Métropole Hotel on Wednesday, March 8th, on

which occasion I hope that, in accordance with ancient custom, each of you will make it not only his pleasure but his duty to attend—and I think we can promise to guarantee you against those grievous pests of medical banquets, a long toast list and lengthy speeches.

In the last century it was the custom to hold the anniversary meeting at twelve noon, for the election of officers; at 2.30 o'clock the Oration was delivered; and, going straight from labour to refreshment, the Fellows of the Society and their friends dined together at four o'clock. Let it be noted that every Fellow was expected to come to the feast; each one had a notice sent to him, and only if he could *not* attend was he asked to communicate with the Secretary.

The mention just now of the name of Lettsom carries me easily on to the second part of my task, and suggests that I cannot do better with regard to it than direct special attention to an elementary part of our Society which he deemed not only of great but of essential importance: I refer, of course, to our Library. As every Fellow of this Society knows, we owe our foundation, and the vigour and prosperity which we now enjoy, to the devotion, foresight, and liberality of Lettsom, who was from the first keenly interested in the creation and development of our Library. Ours was, I apprehend, the first medical library ever established, and in those days, when books were comparatively rare and expensive, the collection must have been of the greatest service and advantage to our growing Society. As time went on, however, the Library apparently began to lose some of its attractiveness, possibly because it was not kept up by adequate additions, and perhaps because of the foundation of other societies and other libraries. But under the constant and fostering care of the Honorary Librarian, Dr. Allchin, it has lately undergone, and is still undergoing, great improvements; whilst his able assistant, Mr. Hall, our resident Librarian, is always in attendance, and willing and ready to place his able services at the disposal of readers. Mr. Hall reminds me of the late Mr. Bailey, the Librarian of the Royal College of Surgeons, whose great delight was to see every table occupied with readers, and to have his services constantly taxed in connection with the work of his department. Higher commendation than this I could not offer to a librarian—but nothing less is due to Mr. Hall. Such of our

Fellows who have not tested him in these respects should make it their business to do so.

Amongst the many treasures of our Library are 15 leather-bound volumes of manuscript which formed the diary, or, rather, the common-place book, of the Rev. John Ward, M.A. Oxon., who was vicar and doctor of Stratford-on-Avon from 1662 to 1681. His handwriting is small, and for the most part hard to read; but with the help of a book which was published in 1839 by Dr. Severn, a former Registrar of this Society, it is easy to obtain a good general knowledge of the contents of the volumes. It is probable that they formed part of the library of Dr. Sims, which we acquired by purchase in 1800.

Ward took his Mastership in Arts in 1652, and I think that I see him sometimes leaving his divinity lectures to work at natural science and medicine. At any rate, as soon as he was able to do so, he came up from Oxford to the "Bell," in Aldersgate Street, so as to be near the "Barber Chyrurgeons' Hall," in Monkwell Street, in order to study practical anatomy under Dr. Scarborough, the friend of Harvey, who gave demonstrations there upon the bodies of persons who had been executed for crime—for, by a charter of Henry VIII, the Barber-Chyrurgeons were entitled to receive each year a certain number of bodies for this purpose. Not very many years ago it was, as we know, quite usual for the professions of divinity and medicine to be combined in the one individual, and it certainly is to the advantage of our profession that in England the custom no longer obtains. But I fear it must be admitted that a class of persons still exist in whom the survival of the dual office lingers—of vicars and vicaresses who cannot keep themselves from dabbling in medicine and quack remedies. More than one country village still contains such unorthodox practitioners, who dispense medical advice to the peril of their too-confiding parishioners, and not infrequently to the annoyance of the local doctor. But let it be clearly understood that the Rev. John Ward was not an empiric of this sort. Though he would be out of place to-day, history shows that he had worked diligently to acquire a knowledge of our profession, and I feel sure that he was well fitted to practise it, under the license which he doubtless received from his Bishop, at the same time that he entered upon his clerical duties. In his diary Ward asked himself specially to remember that he must also "studie

such diseases as are peculiar to women, and also to children so as to be ready at them when I come into the country." He evidently meant to be a good all-round man.

But the point to which I am coming is this, that, taking up his work in Stratford-on-Avon in the 46th year after Shakspeare's death, he must, both as vicar and doctor, have been told of many facts concerning the bard by those who had been personally and intimately acquainted with him. Unfortunately he does not say much about him; but what he did say in these memorandum-books has been already seized upon by the industrious Halliwell Phillipps, and duly recorded in his 'Life of Shakspeare.' Here is one of the extracts, which, coming as it did from a highly educated and learned man who had lived in the village of the Shakspeares, is of undoubted biographical value:—

"I have heard that Mr. Shakspear was a natural wit, without any art atall; hee frequented the plays all his younger time, but in his elder days livd at Stratford and supplied the stage with two plays every year, and for that had an allowance so large that hee spent at the rate of £1,000 a year"—

which in those times was a very large amount, corresponding, perhaps, to £3,000 or £4,000 sterling of to-day.

One other extract I will give, and will pass round the duodecimo volume that you may read it for yourselves just as Ward wrote it nearly two centuries and a half ago—it is a quarter of the way down on the right hand page where the book is opened:—

"Shakspear, Drayton, and Ben Jhonson had a merry meeting and, itt seems, drank too hard, for Shakspear died of a feavour there contracted."

In those times it was customary to call obscure diseases "feavours," just as they are now called "influenza," or "neuroses"; but, whatever was the actual cause of Shakspeare's death, it is evident that local tradition ascribed it, rightly or wrongly, to the effects of that "merry meeting," and it is a matter of satisfaction to us to know that this interesting and important memorandum is in the possession of our old Society. Yes, no doubt that was a "merry meeting." Just think of those three friends together—Shakspeare, Ben Jonson, and Drayton! A pity that Ward could not have been there unobserved with his notebooks!

Time permits me to make but a brief reference to the medical

and surgical jottings of the diary; Dr. Severn has, however, conveniently collocated them in his book, and, considering the period at which they were made, some are highly suggestive and advanced. Thus, he alludes to a man at Cambridge who cured ague by making injections into the veins "as Mr. Wren did into the veins of a dog." What was the drug, I wonder; was it a solution of some preparation from willow bark, or of some metallic germicide? We are now only just beginning to realise that by intravenous injections powerful results may most rapidly be obtained, as with the hot saline infusions in the collapse after diarrhœa, cholera, and hæmorrhage, and in the washings out of the entire vascular system in septic conditions. In syphilis, corrosive sublimate solutions are being employed in like manner; and who could say but that the successful attack of the mean germs of tubercle may be at last secured by some such treatment? Ward says that it was a "fellow at Cambridge" who was working on these advanced lines, and he would be greatly interested to hear that there may still be found at Cambridge, Fellows and others who are doing great work in connection with laboratory experimentation and the advance of medical education generally—Gaskell, Kanthack, Michael Foster, and their associates. Ward was not satisfied with Oxford as a Medical School; at any rate, he left it to come to London for his clinical education, and he quotes Sydenham to the effect that "one had as good send a man to Oxford to learn shoemaking as practising physic." In the 17th century as to-day the first essential for the successful teaching of the art of practising our profession must be the command of clinical material, and in this respect no town or city in the kingdom can ever equal London—yet southern students wander northwards. Possibly Ward thought the Oxford system of practising medicine in those days was too speculative. This is what he writes:—

"A schollar att Oxford applying himself to an elderly physitian of that university, to know whether hee was in a consumption or not, hee askt the schollar, whether hee spitt blood or not? he answered negatively; then said hee, 'tis but a ptysick cough, and I will warrant you from a consumption'; but three months after, his body went to the wormes."

Our clerico-medical diarist took a lively interest in practical pharmacy, and said that it would be an excellent way for

physicians to make up all their physic in troches. In this respect also his thoughts were in advance of his time. It could not be done whilst medical men charged for the medicine which they supplied; but at the end of the 19th century pretty nearly every combination of drugs is made up in troches, cachets, tabloids, or varnished pills, and, kept in stock at the stores, may be bought by the handful for a few pence. Subcribers can choose them for themselves by the list-numbers, and if the hard or varnish-coated drugs pass undissolved through the alimentary canal, at least they have done no harm. In the diary, constant reference is made to small-pox and its ravages, but in the face of the "tremendous experiment" which it has just pleased the Government to institute with regard to this loathsome disease, I have not the heart to make further allusion to it. Government has, indeed, sown the wind; but when the reaping of the whirlwind comes, the plentiful harvest will unfortunately be composed of the innocent, helpless, and ignorant—the very people whom a civilised government are most bound to protect. Our illustrious Jenner was, you may remember, the last recipient of the John Fothergill Gold Medal: could it be that the appreciation which the Medical Society of London showed of his work in 1803 was but the hysterical adulation of incompetent judges? Or was it not, rather, the deliberate expression of opinion of medical men who had known England unvaccinated and pock-marked, and had witnessed the magic change which his beneficent discovery had wrought!

Ward insisted on the need of fresh air for children; he advised the scraping of chronic ulcers, and he gives a graphic account of subcutaneous tenotomy, in the instance of a mountebank who "cutt wry necks, by making a small orifice with his launcet and lifting up the tendon for fear of the jugular veins; then, by putting in his incision knife and cutting them upwards: they gave a great snapp when cut. . . . . When he hath cut it he bends the child's head the other way, and so, by constant bending, itt becomes straight." Had we known more of the treasures of our Library, we should not have regarded subcutaneous surgery as altogether a modern invention. The method has now had its day, however, and is rapidly giving place to a less speculative treatment.

Is every Fellow of this Society aware of the fact that ours is

a lending Library, and that he can look over our catalogues or our shelves and, if so it please him, take home with him an armful of books for reading or reference? But if this is in his knowledge, is he also aware of the fact that on the top of his load he may carry away some of the most recent medical publications, the cloth covers of which are still damp with the binder's glue? In other words, our Council have recently become subscribers to Lewis's Medical Library, so that if the Society is unable to become possessors of every new book, the Fellows may nevertheless have the opportunity of seeing it without delay, and, as I say, of taking it home for reference. This is likely to prove a great convenience, and the Council hope that Fellows will show their appreciation of it by suggesting what books should be sent in, and by making full use of them. Our subscription gives us the right to twenty volumes, which may be changed on the 1st and 15th of each month. On the receipt of a post-card Mr. Hall will insert in the suggestion-book the title of any work which a Fellow may need, and will subsequently inform him (if it is not already in our Library) when it has come from Lewis's.

Even those who have but a slight acquaintance with the early history of this Society are familiar with the name of Dr. James Sims, who occupied the Presidential Chair for 22 years in succession. It was he who from the first had been the great law-maker of the Society, but he found it just as easy to break the laws as to make them. A hundred and ten years ago, when he was beginning that long reign, he was reported to the Council for having taken a pamphlet from the rooms of the Society without leaving regular notice thereof "as expressed in the laws." He was accordingly fined to the full amount, which he duly paid, returning the book. It was Jacob Rayer, the caretaker of the Bolt Court premises, who gave the information regarding Sims's irregular conduct, and in the following year Rayer had again to report him for retaining two copies of the 'Monthly Review.' Sims said that he had lost one of the books; but the Society made him buy a copy to replace it, and fined him for the other. Possibly Sims found it cheaper to "lose" that book than to pay the accumulated fines. Of course, I do not relate this little bit of history with approval of Sims's behaviour, but to show that the Library was freely used and well cared for in those days, and that, having laws, the Society enforced their observance without fear or favour. From

time to time I go into our Library and examine the monthly reviews and journals alluringly spread upon the table, but I regret to say that I never see one which I am for a moment tempted to purloin or to take away and "lose." Perhaps the fault is mine; but I cannot help thinking that the cheapness of paper and printer's ink, and the inconvenient growth of medical journalism, have led to such a watering-down of literary talent that there is no inducement to risk being fined. Would it not be a delight if for one whole year no medical book, review, or journal should be published? And what a happy period of scientific progress it might be made! (In connection with the subject of the abstraction of books, may I be permitted here to make the confession that the proudest moment of my life was when the late Mr. Bailey told me that a book of which I was the humble author had been purloined from the College Library? Would it, thought I, be possible for an intelligent reader to pay a writer a higher compliment?)

With Societies, as with individuals, age brings tolerance; but a century and a quarter ago the Medical Society regarded diffuse communications with intense horror: they would not have them at any price. Even Lettsom had his manuscript on "Intussusception" taken back to him for abridgment. And if such treatment of communications was desirable when the Society was small, and when there was sometimes a dearth of communications, it is urgently necessary now. Let me beg, therefore, that everything brought before the Society be short, sharp, and to the point. Our Society was always anxious that the papers read before them should be discussed. And to see that they had the right sort of material for their meetings, the Council used to read through every communication before sanction was given to its being brought before the Society. For obvious reasons this desirable course can no longer be pursued, and responsibility in this matter now rests chiefly with the Honorary Secretaries. It is intended, however, during the coming session to relieve the Secretaries of much of this inconvenient responsibility, by more generally referring proposed communications to the various Committees of Reference. In the infancy of the Society the procedure in connection with each paper was as follows:—The paper was read by the Council, and, if approved, was ordered to be brought before the Society. On the first

occasion of its being read before the Society, however, it was not discussed, but on the conclusion of the reading a vote was taken as to whether it should be publicly read a second time. If this was declared in the affirmative, the discussion took place after the second reading, and a vote of thanks was then passed to the reader. It is impossible to go back to that plan, but we must all feel that it had much to commend it. The Fellows of a Society derive more benefit from the full discussion of a few papers of high merit than from hearing many papers of lesser value. There is one peculiar way in which societies suffer:— A gentleman has a manuscript of, let us inoffensively say, *x* value which he greatly wants published. He has tried by various methods to get it into print, but without success; and at last, by the help of influential intercession, he induces the officials of one of the Societies to accept it. Let me say, with all politeness, that it will be no use his bringing such wares to Lettsom's Society during the coming session; I had rather that we failed to have material for one of our Mondays than that the Society should be thus exploited.

In the Oration which I had the honour of delivering before this Society about 18 months ago, I made a suggestion, in all seriousness, that it might be advantageous if we set apart one meeting in the session for the discussion of unsuccessful cases, or those which had ended in disaster. I now ask how it would fall in with the views of the Society to make the last meeting in this year a "Surgical confessional evening"? I do not know if my own experience differs widely from that of other surgeons, but I am the unhappy possessor of considerable wealth of clinical material for such an evening, which, in the silent watches of the night—and at other times—is apt to thrust its records before my inward eye. I would be willing, therefore, to start the business of the evening. There would, of course, be no report of that meeting; possibly, indeed, the reporters would themselves like to bring forward a case or two of their own. At any rate, I should be glad to hear what my surgical confrères think of the idea.

In conclusion, permit me to make a request of each one of you, namely, that you specially endeavour to bring recruits into the Society. We want particularly young men, though, of course, we shall gladly welcome those who can no longer claim the

privileges and advantages of youth, for we have no age limit. But young men are those who are going to do and to carry on the work of the Society, so we must have a fair proportion of these recruits. As you well know, you can safely advise your friends to join our Society whatever their line of practice is going to be—medical, surgical, obstetric, general, or special. In our rooms they will make the acquaintance of men in all lines of practice, and they will soon find acquaintanceships ripening into friendships. At our meetings they will hear all subjects treated of in turn, and discussed in a bright and practical manner; I am glad to say that the discussions are sufficiently informal as not to create weariness or inspire awe. Lastly, the annual subscription is small, and it entitles one to a copy of the 'Transactions' of the Society—a work which seems steadily to be increasing in value and interest. But beyond all this, and beyond the various and manifold benefits offered by the Society, the working Fellow will discover advantages in our Library which, considering the smallness of his annual payment, cannot be offered by any other Society in London and probably are without equal in any other city in the world.

May this, the 126th year of the useful life of the Medical Society of London, be as happy as any one of those years through which it has already passed; may there be no falling off in practical value of the work done in it; and may the spirit of tolerance and fraternity which Lettsom breathed into that little group of medical friends in the month of May, 1773, continue to increase amongst our Fellows, not only for the session which is now beginning, but until time itself shall be no more.

SOME CONSIDERATIONS PRELIMINARY TO THE STUDY OF DYSPEPSIA.

By W. H. Allchin, M.D., F.R.C.P.

Gentlemen,—I take it that I shall meet with but little opposition if I assert that there is no department of practical medicine or group of clinical phenomena which are investigated, described, and considered with a greater vagueness and looseness of thought and method than the subject of indigestion. It is not that there is in the diseases of the circulatory apparatus, of the respiratory organs, or even of the nervous system less of which we are ignorant, but in connection with these and other functions what we do know is tolerably clearly defined from what we do not know, our ignorance no less than our knowledge is formulated, and, so to say, we know where we are. Next to positive knowledge, a clear recognition of what is not known is the most valuable aid to the progress of any branch of inquiry, and particularly so in one of so practical and complex a character as clinical medicine. In respect, however, to the maladies dependent upon errors in digestion, a singular lack of precision in thought and language appears to be all but universally prevalent. Nor does this seem to be quite justifiable, since on the one hand our knowledge of the normal physiology of digestion is as well advanced as is that of the other functions—more so, indeed, than of some—and on the other hand the frequency with which digestive disturbances occur offers the fullest opportunity for study if pursued on lines which have proved so fruitful in other directions of pathology.

I would at once desire to disclaim all pretension to an accuracy that is not possessed by others, but I am merely stating as a fact what I believe would be at once admitted; and for example of my contention I would refer to our text-books and systems of medicine, and ask any one to compare the mode of handling the sections devoted to diseases of the digestive organs with those of other functions. In the latter we have for the most part an attempt at logical treatment of the subject. The causes, symptoms, morbid anatomy, and other aspects of the several diseases are treated on

a more or less uniform plan; facts are stated, provisional hypotheses are recorded, and a coherent and sequential view of each disease or group of diseases is presented, from which the enquirer may learn what is known and perceive the directions in which further information is required; in other words, there is an orderliness in the treatment of the subject which has been the outcome of systematic observation and scientific uniformity of plan. But if we turn to the chapters which deal with disorders of digestion or the diseases of the organs of digestion, there is at once apparent a departure from the method which obtains elsewhere, and confusion takes the place of order; the causal relation of symptoms seems to be imperfectly grasped, the limitations of the digestive process as distinct from absorption and other factors of nutrition are disregarded, and the manifestations of structural disease in the organs concerned are mistaken for the evidences of errors in the chemical changes involved.

Again let me say that my presumption does not extend to asserting that I am prepared to set the whole question straight; but if I can first of all gain your agreement that I have fairly stated the case, it will be a step towards the rectification of the position and dispose you more favourably to consider this my contribution thereto.

It is a poor pursuit, I am fully aware, finding fault with the language of any branch of natural science. It is inevitable that as the science progresses, the denotation of the terms and significance of its nomenclature must vary, until it is only by arbitrary consent they come to possess an agreed-upon meaning. And this difficulty doubtless is met with in no small degree in respect to digestion disorders. But there would seem to be a reluctance to employ many words in their legitimate sense, and still more to use them with any consistency. I shall make my position clearer and render your criticism of my remarks the easier if I set forth the sense in which I employ some of the words I use, and this will be no peculiar or special use of them, merely such as they legitimately carry.

And first let me say that to my mind the very word digestion, and still more its correlative indigestion or dyspepsia—for they are synonymous—are apt to be employed with much uncertainty or at all events variability in their range of meaning. That digestion is a function of the body concerned with the food, and

upon the maintenance of which the bodily health depends, is generally accepted; but of the scope of the function most different opinions appear to be entertained by medical men, with corresponding confusion as to the real nature of its disturbances. Surely it is advisable in this as in other functions to follow the physiologists in regarding digestion as a term denoting the entire series of changes which the ingesta undergo during their passage along the alimentary canal from their entrance in the mouth to the expulsion of the undigested, and unabsorbed residue. If this be the proper meaning of digestion, it is obviously improper to restrict the idea of it to those changes which occur in the stomach only, and if it may be said "Surely no one ever takes such a view," I can only say that in the practical application of our physiological knowledge to disease, the word indigestion or dyspepsia is constantly so limited. "Dyspepsia is a general name which is generally used as synonymous with indigestion," says a distinguished member of our profession in a standard work recently published. "It properly means discomfort and pain during digestion in the stomach, but it is frequently used to signify discomfort or pain during the process of digestion whether in the stomach or intestines. The vagueness of its significance has both advantages and disadvantages. The disadvantages of the name are that it signifies so many different conditions, that it is little or no guide to the exact functional or organic lesions of the several organs concerned in the digestive process; and without a more or less accurate knowledge of the nature of a disease we cannot hope for success in treatment. Its advantages, on the other hand, are that it includes a group of symptoms which cannot always be referred to an exact physiological and anatomical basis. In this respect it corresponds to albuminuria or jaundice, affections which, although they can often be assigned correctly to their causes, yet sometimes defy exact diagnosis."* To my mind a more unscientific justification for the use of a word in a wrong sense is scarcely to be imagined. Its use is advocated on the ground of ignorance, which I would contend this very use helps to increase. But the quotation illustrates the current view that indigestion is essentially

* Allbutt's 'System of Medicine,' vol. iii, art. "Dyspepsia," by Dr. Lauder Brunton, F.R.S., p. 385.

a stomach affection, and imperfections in the entire range of processes in the mouth and in the intestines are relegated to a wholly subordinate position, scarcely considered in fact. Now of the four successive stages of digestion, the oral or salivary, the gastric or peptic, the pancreatico-biliary and the intestinal, following one after another as they do, it is probable that the gastric is far from being the most important, its deficiency being made up for at other stages.

The simple object of the process of digestion, and so far as we know all that is attained by it, is the conversion of certain ingredients of the food into a fluid and diffusible condition whereby they may be absorbed into the blood or lymph currents, they of themselves as taken being wholly or almost wholly unfit to be so absorbed without undergoing digestive changes. I say "almost wholly," for there is some reason to believe that a very slight degree of absorption can take place even of those substances which ordinarily undergo considerable digestive change, as, for example, the beneficial results following nutritive enemata. It is the proteid, fatty, and carbohydrate elements of the food which specially require digestion, and of these the first-named are chiefly acted on in the stomach, the salivary digestion of the starches and the splitting up of the fats proceeding only to a very partial extent in that organ. But although the stomach is thus mainly concerned with proteid digestion, it is to be observed that the pancreatic secretion is equally if not more capable of effecting proteid conversion into peptones, and what is of equal importance to the present questions very little, if any, absorption of the digested proteids takes place from the stomach. Without going so far as to say that under ordinary circumstances the stomach is superfluous, physiological considerations would prepare us for the statement that its functions may in great measure be compensated for elsewhere in the canal. Experiments on animals have gone far to show that this is so, and the recent remarkable case of complete extirpation of the stomach recorded by Dr. Carl Schlatter* has conclusively proved that the stomach is not an essential organ, inasmuch as the patient within a few weeks of the operation was taking without discomfort an ordinary dietary consisting of meat, bread, eggs, milk, &c., and a careful investigation of the metabolic products of the patient as compared

* 'Lancet,' January 15th, 1898.

with the ingesta, conducted by Hoffmann in Eichhart's clinic,* demonstrated a state of complete nitrogenous equilibrium, the patient gaining weight. The particular value of the stomach as a receptacle for receiving the food and passing it on into the intestine, portion by portion, and the use with which the hydrochloric acid of the gastric juice is credited as a guardian from bacterial infection and a controller of putrefactive fermentation in the intestine, are alike discounted by the history of this extraordinary case. Surely, then, in view of this quite subsidiary action on the part of the stomach, it is quite indefensible to regard the gastric digestion as the most important stage in the whole process, and that dyspepsia of other than gastric origin is practically non-existent. I feel that the preliminary step to a more rational understanding of indigestion is so to enlarge our views of the comprehension of this term as to make it coterminous with digestion proper, and recognise the occurrence of real duodenal and intestinal dyspepsia, more frequently than we are in the habit of doing.

Again, it is most desirable that the conception of "digestion" should be strictly limited to the process as above defined, and that it should not be taken to include the function of absorption. This is wholly different in its nature from the complex chemical changes which constitute the former; and whilst it is the second stage in the progress of the ingested food-stuffs towards the nutrition of the tissues, there for the first time are the digesta brought within the range of living protoplasmic activity as they traverse the columnar epithelial cells of the intestinal mucosa. The two functions have little or nothing in common in their dealing with the nutrient material, and as the physiologists consider each separately yet successively, so should the physician refrain from confusing the two, and endeavour, so far as may be, to ascribe to each the various symptoms which his dyspeptic patient may present. I am fully aware that defects in the due absorption of the digested material may become a determining cause of indigestion itself, just as an imperfect digestion of ingesta may interfere with their proper absorption; but this interrelation does not justify the processes being confused, or their limits disregarded.

There are other points also connected with the exact scope which the term digestion implies, which, to judge from the

* 'Munch. med. Woch.,' May 3rd, 1898.

general character of writings on the subject, are often forgotten, especially when it is a perversion of the function which is under consideration; and one of these is the relation which the process bears to the digestive organs. This is peculiar, and has no counterpart in the animal economy. The function of digestion is a combined motor-secretory process, and the organs by which it is performed are muscular and glandular, the effective agents of each being specialised protoplasmic cells. The food-stuffs are propelled along a lengthy tube, and mixed with the secretions by muscular contractility, and the secretions successively perform their physico-chemical effects on them in virtue of the enzymes and acid or alkaline substances which they contain. But the point is that these changes take place outside the tissues, although in the body. We see nothing like this in the other functions whereby the bodily nutrition is maintained—absorption, circulation, respiration, and excretion—and still less in the activities of the nervous system. As productive of disease there are, therefore, two very different conditions to be observed: on the one hand, disorders of the secreto-motor apparatus—structural, nervous, or vascular—by which the functional capacity of the apparatus may be interfered with; on the other, errors in the physico-chemical change taking place in the digesting material. It is true that these necessarily react upon one another, that imperfections in the digestive process, due, may be, primarily to errors in diet, may determine structural disease of the digestive organs, just as this latter will lead to mal-digestion. But it is very desirable to recognise their essentially distinct character, and the fact that each may exist to a certain degree independently of the other, in the production of what are known as dyspeptic symptoms.

One advantage of having this aspect of the subject in mind is that it furnishes a rational basis for classifying the causes of indigestion.* This subject I have elsewhere treated of, and it is sufficient for my present purpose to mention that the immediate causes of dyspepsia are:—

(1) *Errors in diet*, whether of quantity, quality (including irritant and toxic substances), or in the form in which the food is presented.

* "On Some Relationships of Indigestion." Hunterian Society's Lecture. 'Lancet,' October, 1897.

(2) *Diseases of the digestive organs*, whereby their motor or secretory functions or both are impaired. Such diseases are:—

(i) Structural—inflammation, degeneration, new growths.
(ii) Impaired nervous control, whether of central or reflex origin.
(iii) Abnormal blood and lymph vascular supply.

(3) *Improper bacterial action* in the gastro-intestinal contents.
(4) *Defective absorption* of the digesta.
(5) *Abnormal intestinal excretory processes.*

I am not concerned now to discuss these causal conditions, upon each of which much might be said, even of the last-named, which I think promises a not unfruitful direction of inquiry.

But I would point out that whilst all these abnormal states produce in a greater or less degree disturbances in the physico-chemical processes which take place in the gastro-intestinal contents, and as such are the cause of dyspeptic symptoms, the second of these, viz., the diseases of the organs, may give rise in addition to symptoms and signs expressive of these diseases —for instance, malignant disease of the stomach, besides the dyspepsia it may determine by its interference with the motility of the organ and the grave alteration it causes in the composition of the gastric juice, may lead to hæmatemesis, gastro-ectasis, &c., which are not evidences of indigestion, though due to diseases of the digestive organs. And further, inasmuch as the very process of indigestion may and does set up disease in the organs, certain of the complex symptoms which a patient presents are referable to this secondarily induced morbid state. Theoretically, therefore, these causal conditions give rise to disease manifestations in these different ways: first, dyspepsia proper by mal-performance of the digestive process; and secondly, the symptoms of diseases of the digestive organs, which may be primary in origin or secondary to the existing dyspepsia.

I pass now to say a few words in general upon the symptoms of dyspepsia, conditioning my remarks upon the nature of indigestion as I have set it forth, and the two-fold origin I have just described. It is customary—and indeed I think no self-respecting writer on the subject would feel at liberty to refrain—to describe the symptoms of indigestion as "protean." I am not aware that the term tends to elucidate the problems at issue, but having

conformed to established ways I proceed to express the opinion that the actual clinical manifestations of dyspepsia are not so numerous as is often supposed; that many are rather evidences of the condition to which the dyspepsia is due; that others are such remote results as to be practically removed from the exciting cause; and the remainder, which are directly to be associated with flaws in the digestive process, and therefore properly dyspeptic, are capable of being grouped under very few heads, which much facilitates their comprehension.

Although not strictly definable, it is none the less convenient to speak of them as local and remote, the first being obviously and directly connected with the digestive organs. They may be tabulated thus:—

A.—Local.

I. *Subjective Sensations.*

Discomfort. Weight and oppression. Sinking.
Pain.
Nasty taste in mouth.
Disordered appetite.
Acidity. Heartburn.
Nausea.

II. *Objective Phenomena.*

Eructations.
Vomiting.
Flatulence.
Diarrhœa. Constipation. Abnormal stools.

B.—Remote.

Nervous.—Headache. Vertigo, giddiness.
Subjective affections of sight and hearing.
Drowsiness, heavy sleep, torpor, wakefulness.
Mental irritability or depression, hypochondriasis, melancholia.
Referred pains. Cramps.

Circulatory.—Palpitation. Cardiac irregularity. Intermittence. Syncope.
Cardiac pain. Angina.
Flushing of face. Coldness of extremities.

Respiratory.—Cough. Dyspnœa. Sighing.

Cutaneous.—Various urticarial and erythematous rashes.
Urinary.—Deposits of urates, phosphates, oxalates, uric acid.
Albuminuria. Glycosuria.
Presence of aromatic sulphates.

Such a list—and I do not pretend that it is absolutely complete—is, I admit, a long one, and as varied in manifestations as it is lengthy. But it is not this multiplicity of signs and symptoms collectively denominated dyspeptic which, to my mind, makes their investigation difficult, so far at least as their reference to cause, and hence diagnostic value, is concerned, but rather the fact that many totally different conditions give rise to similar symptoms. The aim and object which, as clinical observers, we set before ourselves when investigating a case or group of cases of disease is, first of all, by systematic procedure to ascertain as accurately as may be, with various instruments of precision and otherwise, the departures from the normal working of the body, and such alterations in gross structure as physical examination may disclose. Such evidence—the signs and symptoms of the disease—we proceed to consider in respect to their underlying cause, and from the evidence infer what is actually wrong—what, in fact, is the structural abnormality upon which the functional defects depend. In the course of making this inference or diagnosis we rely first and mainly upon our knowledge of the normal organism in its structure, gross and minute, and in its action; secondly, upon the experience of what structural or organic changes have been discovered previously to be associated with certain signs and symptoms. This is the basis of procedure upon which we teach our students, and more or less consciously it is the plan we pursue in every patient we examine, however much we may jump to conclusions. It is in the application of this principle to the causal diagnosis of dyspepsia that the real difficulty in the problem lies, the fact that no single symptom is diagnostic—I will not say pathognomonic—of the cause, but that, as I have said, the same symptom may be expressive of various underlying states. Take, for instance, the question of pain; how can that help us to ascertain the nature of the cause in any given case of indigestion? and to do that is essential before any rational plan of treatment can be proposed, to avoid the unscientific routine of bismuth, acids, alkalies, bitters, pepsin, &c. Reference

to the list of causes I have given will suffice to show that we must seek to differentiate between errors in diet and morbid states of the digestive organs; we may for the moment disregard the other and rarer causes of mal-digestion. This is the first and fundamental step in the procedure. A moment's reflection will serve to show that pain may follow something that has been taken into the alimentary canal, whether such an irritant as a metallic poison or some indigestible article of food. On the other hand, so distinct a structural lesion as an ulcer of the stomach or a growth in the intestine may be provocative of considerable pain quite apart from any special irritation that food may induce. Or again, in conditions of the stomach where the actual disease is less obvious, the condition which is known as gastralgia, when the sensory nerves of the viscus are unduly susceptible to the reception of impressions of a painful character—here pain is a marked, and indeed almost the only, symptom. What, then, is the significance of pain as diagnostic of the existence of either of these morbid states? Nor can more reliance be placed upon its character, so inadequately described by the current adjectival terms, or the situation in which it is experienced, nor even always its relation to food, since in well-recorded cases of ulcer, relief has followed a meal—a circumstance that in the main does serve to distinguish a gastric ulcer from a gastric neuritis. Or again, nothing is more certain than that the digestive process may be most imperfectly performed without anything resembling pain being experienced; even a gastric ulcer has been found *post mortem*, the presence of which has not been suspected during life; whilst pain is commonly absent from many cases of defective motility of the gastro-intestinal musculature; and also where there is good reason to suspect secretory imperfections as a cause of mal-digestion; and serious dietetic errors may be long continued without more than the slightest sensory discomfort. It follows, then, that the presence or absence of pain cannot be taken as definite evidence of indigestion, far less as indicating with any approach to certainty the cause to which an indigestion is to be attributed. And what is true of so pronounced a symptom as pain is also true of other dyspeptic manifestations; vomiting and constipation, for instance, are each referable to food, to obvious structural disease of stomach or bowel, or to nervous causes acting centrally or reflexly. And the same may be

affirmed in respect to the remoter symptoms, which require first of all to be clearly recognised as dyspeptic in origin before the particular factor of the dyspepsia can be positively indicated—such, for instance, as headache, anginous attacks, cough, deposits of urates in the urine, oxaluria, &c.

The main difficulty, I repeat, which attends the clinical diagnosis of dyspepsia is not the detection of the symptoms and signs, which, for the most part, are obvious and easily ascertainable, but it is the association of these symptoms when perceived with the morbid condition which determines them.

The difficulty, also, is further increased by the very small part that physical examination plays in the detection of disease of the alimentary viscera. Excluding the liver, an organ whose connection with digestion proper is far less important than formerly supposed, we really learn very little by the application of our senses of sight, touch, and hearing. Certain alterations in the size and situation of the stomach, abnormal distension of the colon and a proportion of new growths connected with the organs in question may be recognised; but if we compare the range of application of these methods with what can be done in connection with the heart and lungs the disadvantage at which we are placed is at once obvious. To a very great extent we rely for our recognition of primary disease of the digestive organs upon the presence of symptoms of indigestion, symptoms which are equally indicative of condition in which the organs are little if at all affected.

And here it will be necessary for me to refer to the clinical varieties of dyspepsia as they are described in books and elsewhere.

So long as indigestion was considered to be a malady mainly, if not entirely, connected with the stomach, such forms as "irritative dyspepsia," "atonic dyspepsia," and the like, found a place if not a precise meaning; the phrases suggested a corresponding plan of treatment, by alkalies or acids, sedatives or tonics, by the success of which their use was justified. Clearly such names should be discarded now, and as surely should those based upon the preponderance of one symptom, such as "flatulent indigestion." Whilst the continued use of such a term as "biliousness" or "bilious dyspepsia" can only tend to obscure the real state of the case, and may well be left to denote that condition of

uncertain patl ology which, it has been well said, is cured by blue pill. Far less open to objection is a classification based on the site of the digestive position, oral, gastric, duodenal, intestinal. Though the data for such a grouping are still very deficient, it is certainly an end to be kept in mind in the consideration of the subject from a diagnostic point of view. From another and correlative aspect to this the forms of dyspepsia may be grouped on the particular element of the food which is ill digested, such as proteid indigestion, fat indigestion, or carbohydrate indigestion. A complete knowledge of the subject, which we are far from possessing, would enable us to go far in this direction. Probably the most satisfactory plan on which to found a classification is that based on causation—the immediate causation, that is, such as I have described. This arrangement would give the following forms:—

1. Dietetic.

2. Symptomatic, *i.e.*, the manifestation of primary affections of the digestive organs, structural, nervous, and vascular.

3. Bacterial indigestion.

4 and 5. The rarer conditions of indigestion due to defects in the due absorption of the digesta, and to improper excretory processes taking place in the intestine, probably occur so infrequently—at least apart from other causes—as scarcely to require special naming, but the terms "absorptive" and "excretory" dyspepsia are consistently suitable.

The full application of such a scheme would involve the consideration of the locality in which the indigestion is taking place—mouth, stomach, or intestine; and would also of necessity take note of the kind of indigestion, whether proteid, fatty, or amylaceous. But whatever plan be adopted, the correlation of symptoms and cause will still remain a desideratum, and until that has been worked out any arrangement can only be provisional. I would, however, claim for the one proposed a nearer approach to scientific precision than any in current use.

Not the least among the advantages that would follow, as I conceive, the adoption of some such plan as this would be to furnish a clearer conception of the relation of the several diseases of the digestive organs to indigestion, and to doing away with such meaningless, and I would add mischievous, terms as "functional disorders" of the stomach or bowels. All disorders of the

organs concerned in the digestive process which interfere with the proper performance of that process are functional disorders as much as they are structural diseases. The existence of the one implies the other. The deficient motility and altered secretion which characterise an acute gastritis or a gastric cancer are as surely the causes of indigestion as the less obvious, but none the less existent structural change which determines the motor secretory imperfections in so-called "gastric irritation" and "gastric inadequacy," or even "nervous dyspepsia," which may be taken as examples of "functional disorders of the stomach." How far these conditions can exist without departures from the normal structure of the organs concerned, however slight and transient these departures may be, I confess I am at a loss to understand.

"Gastric irritation," says Dr. Sidney Martin,* "may be defined as a functional disorder of the stomach in which there is irritation of the organ, produced by the food itself in the form in which it is swallowed, or by the food in the process of digestion; it may be primary (*i.e.*, occur in healthy individuals), or it may be associated with or pre-disposed to by certain diseases or general conditions of the body. Food bears to gastric irritation and to gastric catarrh the same relation that irritating particles and gases bear to bronchial irritation and bronchitis." I should myself have thought that bronchial irritation was as much a structural imperfection as bronchial catarrh or bronchitis, and, *mutatis mutandis*, gastric irritation as gastric catarrh, and that the altered function (disease) was as much an expression of altered structure in the one as in the other. I have on a former occasion† criticised the expression "functional disease," and need say no more, except that if there ever exists a condition in which the digestive organs are quite healthy, and the secretions and motility of the canal normal, and yet, owing to the nature of the food, some maldigestion of that food takes place without the organs or their functions being affected, then there would be a purely functional indigestion unaccompanied by structural defect; though how long such a condition could last, or whether it could be recognised, or whether, indeed, it actually could occur, I am not prepared to

* 'Functional and Organic Diseases of the Stomach,' by Sidney Martin, M.D., F.R.S., 1895.

† 'Westminster Hospital Reports,' vol. ii, 1886, "Functional Diseases."

affirm; but that is an entirely different thing from a functional disorder of a viscus.

The bearing of the foregoing remarks, which appear to me to be among the necessarily preliminary considerations to a study of dyspepsia, would, perhaps, be better appreciated were I to take up somewhat in detail one of the conventional varieties of indigestion; and no one would serve my purpose so well as the so-called "nervous dyspepsia," a term that is more frequently on our lips than, I fancy, its real understanding warrants.

Most authors follow more or less closely the description of Leube, who nearly 20 years ago published a paper* on the subject, and in so doing gained the credit of being the first to differentiate it from other forms of chronic dyspepsia. The causal basis of his definition of the condition is the existence of such a general nervous state as hysteria or neurasthenia; and practically it comes to this, that dyspeptic symptoms occurring in such patients, with the singular proviso that the digestive act, so far at least as the stomach is concerned, is accomplished within the normal time as ascertained by a test meal, constitute nervous dyspepsia. This latter circumstance is an essential feature of nervous dyspepsia according to Leube, but it is very doubtful whether such a position can be maintained. Apart from that the reference of the condition to a fundamentally underlying nervous cause is satisfactory, though not, perhaps, wide enough as originally laid down. Dr. Sidney Martin, who from the point of view of stomach only, adopts in great measure Leube's view, also says †: "There are many cases of gastric irritation, and, indeed, of other disorders of the organ (gastric insufficiency), in which certain symptoms attain such prominence as to give a feature to the particular case. Such symptoms are without exception referable to some change in the nervous system, and are by some included under the terms nervous dyspepsia and neurosis of the stomach." In this case the limitations of the malady are conditioned by the symptoms rather than by the cause. Or again, Dr. Goodhart tells us that "although there are all sorts of different dyspepsias elaborated out of the heads of individuals, if I were going to write a book on indigestion I should first devote

* "Ueber nervose Dyspepsie," 'Deutsch Archiv f. klin. Med.,' 1879, vol. xxiii.

† *Loc. cit.*, p. 208.

myself to a volume on diseases of the nervous system. Surely," he continues, "I cannot be wrong when I make the statement that of the number of dyspeptics that fill the doctors' lists the great proportion by far are neurotics in one form or another. It is really no great exaggeration to say that there are only two forms of indigestion; that produced by over-eating and drinking, and that due to failure of nervous power. There are other conditions that are met with as rarities, but these who knows anything about? Then we talk about acid dyspepsia and atonic dyspepsia, as if any dyspepsia were other than acid, and from my point of view as if atonic dyspepsia were necessarily something different." ("Common Neuroses." The Harveian Lectures, 1891.) With all submission there seems to me a good deal open to question in these statements, but as bearing upon my present purpose, viz., nervous dyspepsia, it is clear that the distinguished writer regards it as a very widely extended malady, far more so than the other authorities I have quoted. Possibly, as his theme was "common neuroses," he had been regarding disease at large through "nervous" spectacles. In marked contrast is the opinion of a later author,* who states that "in nervous dyspepsia there is an increased irritability of the stomach nerves, especially the sensory. Whether this increased irritability is really due to a mild inflammatory process in the mucous membrane is difficult to determine. In the neurasthenic a slight degree of inflammation not recognisable by our present methods could produce morbid manifestations in the form of a dyspepsia, which only an advanced gastritis would otherwise call forth." And he concludes that nervous dyspepsia is a disease by itself, and chiefly a sensory neurosis, though the motor and secretory functions may also be impaired in it; that it is not so frequent a disease as has been assumed; and that though it usually exists along with other nervous manifestations it is wrong to assume it as a symptom of neurasthenia. My object in submitting these quotations—and they are but a sample of many—is to show the extreme diversity of views that is held upon the nature and scope of a malady that we are constantly in practice speaking of—a diversity, I would maintain, that is due to the generally vague and indefinite notions that prevail upon the real meaning of indigestion. Throughout these opinions there runs, more or less

* Rosenheim, 'Berl. klin. Woch.,' November 1st, 1897.

expressed, the idea that indigestion is a gastric perversion only, and in some cases symptoms, and in others underlying nervous states, are made the basis for the separation of the particular variety of dyspepsia. I much doubt whether any not infrequent disease of any other system would be so vaguely dealt with, or that an inquirer would gain such little real information to help him understand the nature of the malady were he to seek for it from any of these authorities I have quoted, whilst the divergencies amongst them would only mystify him.

By the adoption of some such arrangement as I have indicated the place and scope of nervous dyspepsia would be at once laid down, and, what is of equal importance, its relationship to other modes of digestive disturbance. It would be defined as that form of disturbed digestion due to impaired innervation of the digestive organs, whether mouth, stomach, intestines, or associated glands, and leading to imperfect changes in any or all of the different food constituents — proteid, fat, or starchy, according to the locality of the affection. The disordered nerve control would be due to, or, in other words, the causes of nervous dyspepsia would be—

(*a*) Disturbances of central origin.
Emotional states; anxiety; overwork.
Obscure conditions of the nervous system, such as hysteria, neurasthenia.
Gross structural lesions, such as cerebral tumour, meningitis, locomotor ataxy.
Toxic bodies.
(*b*) Disturbances of reflex origin, as from kidneys, ovaries, &c.
(*c*) Peripheral irritation of nerves of alimentary canal.

The term nervous dyspepsia, therefore, would not denote a group of symptoms, but an indigestion primarily of nervous origin.

The function of digestion consists, as I have reminded you, of a series of physico-chemical processes affected by various secretions, and aided by movements which mix and propel the gastro-intestinal contents; and indigestion is due to some flaw in one or more of these different acts, which causes what we speak of as dyspeptic symptoms. Now both the secreting and the motor factors of digestion are controlled by the nervous system; directly

by influences on the muscle and gland cells, and indirectly by regulating the blood and lymph supply to these structures. It would be expected, therefore, that the symptoms of a nervous dyspepsia would be such as are directly traceable to a disturbed motility or secretion, or to both, and such is actually the case; for example, the retching and vomiting that fright or anxiety may produce; the obstinate eructations or vomiting, perhaps after every meal, of the hysteric; the characteristic vomiting of cerebral tumour, or of the gastric crises of locomotor ataxy; or the circulation in the blood and nerve centres of certain poisons. Note also the arrested movements of the stomach which may attend emotional states, as may be seen by the undigested meal vomited long after it should have passed through the pylorus; or the increased intestinal peristalsis, leading to diarrhœa, from fright or other psychical states. These illustrate the perverted movements determined by impaired nerve control. Our knowledge of the innervation of the digestive glands is tolerably precise as regards the salivary and rather less so in respect to the pancreas, while little is known with any accuracy about the nerve governance of the gastric and intestinal tubules, to the activities of which the peristaltic movements of the gastro-intestinal musculature appear to be an important stimulus. We have, however, very distinct evidence that the gastric juice is liable to considerable alterations in quantity and quality, a marked increase or diminution of the hydrochloric acid, and perhaps also, though less certainly, of the pepsin. Such modifications are among the most marked phenomena of nervous dyspepsia, to which many of the more obvious symptoms of that affection are due. The local sensory manifestations are attributable to the disturbed motor secretory functions. It is seldom that severe gastric pain is complained of, and such a symptom, when present, I regard as gastralgia due to a hypersensitive condition of the nerve terminals —a hyperasthenia comparable to that of certain areas of the skin noticeable in gastric ulcer—and though not infrequently accompanying indigestion, is not itself any evidence of mal-digestion proper. Alterations of appetite are among the best known results of many nervous states, and all degrees of absence, excess, or capriciousness are frequent. The marked degree of perversion associated with extreme emaciation, known as *anorexia nervosa*, is due to a more profound disturbance than that of digestion merely;

more probably the absorption of the digesta is defective, or the entire metabolism of nutrition at fault, since it is a wholly different condition from that following simple deficiency or even deprivation of food inflicted on a previously healthy person.

Flatulence, which in my experience is not a marked symptom of this variety of dyspepsia, is rather to be regarded as a secondary result of the improper digestion of amylaceous constituents, except in such cases as it is due to sudden and extensive escape from the blood vessels into the intestine. Passing to the more remote symptoms — headache, dizziness, drowsiness or insomnia, mental irritability, hypochondriasis, irregular pains, cramps, cardiac palpitation and irregularity, coldness of extremities, &c.—it would be exceedingly difficult to say how far such manifestations are not due to the primary nervous disturbance which has been the cause of the indigestion. These and other symptoms of the same character, when they are clearly to be attributed to mal-digestion, are probably caused by the formation of toxic substances in the course of the digestive changes which are absorbed and circulated, or to variations in blood pressure.

The last point to which I would refer is that no one of these symptoms is in any degree diagnostic; each one of them may occur in other forms of dyspepsia in which the nervous influence is not primarily perverted, if, indeed, it is at all. Vomiting, diarrhœa, and other motor disturbances are among the commonest evidences of an indigestion of dietetic causation or of gastritis, as likewise flatulence and the whole series of remote phenomena. Hyperacidity is met with under similar conditions as in gastric ulcer, whilst a deficient secretion of gastric acid commonly occurs in gastric cancer. As I said earlier in my paper, it is that these various symptoms are none of them specially connected with any one cause, but are more or less common to all causes, that makes the ascertainment of the cause by study of the symptoms so difficult—the difficulty, indeed, which characterises the whole subject. But I do not regard such a difficulty as insuperable, though I am not prepared now with the solution; but I feel convinced that before any rational treatment can be propounded with reasonable hope of success, it is this problem which has to be overcome; and as preparing the ground for so doing by an orderly and scientific method on a physiological basis these observations are offered.

Dr. MAGUIRE said he had listened with great interest to the paper, especially to that part of it which touched upon the turmoil into which medical literature had fallen on this subject. During the last 15 years a great deal of time and energy had been expended, especially in Germany, in studying the chemistry of digestion, with much increase to their knowledge of the chemical changes which food underwent during its passage along the alimentary canal. At the same time, he thought they were very little the better for this work so far as the treatment of dyspepsia was concerned. He himself had tried to classify the numerous cases of dyspepsia that came before him—irritative, flatulent, atonic, &c.—and had endeavoured to treat them accordingly, but the result thus obtained had been not much better than that of 25 years ago. He recognised that a proper classification was indispensable, and to assist in attaining this object he recalled the fact that neurologists in nervous diseases unanimously held the opinion that those functions which were the last to be developed were usually the first to go in disease and the last to return, while fundamental functions which were the first to develop were the last to go and the first to recover. He proceeded to argue that this principle, which was recognised in relation to nervous diseases, might also apply to the other system with which they had to deal. Throwing on one side the seat of dyspepsia and its causes, he asked them to look at broad facts from a developmental point of view, and to consider the order in which the digestive functions were developed. The food of the embryo was the pure albumen of the yolk-sac, which required no digestion. After birth, the infant's food was milk containing proteid in the form of casein, carbohydrates, and fats. The only constituent which required digestion was the proteid. The digestion of fat in the ordinary healthy adult did not amount to much more than emulsification. The emulsification of fat in milk was perfect and could not be surpassed, and the carbohydrates also did not require to be digested. Proteid, on the other hand, was digested. It was not absolutely perfect digestion in the infant, as shown by the fact that very slight variations of composition, such as that involved by the substitution of cow's milk for human milk, sufficed to upset the child. Even the slight change entailed by taking in the colostrum corpuscles would often start diarrhœa and dyspepsia. The digestion of carbohydrates did not begin until the seventh month, and was probably not complete until after 12 months of age. The digestion of ordinary fat was probably not developed to any extent until after puberty, and the distaste of the child to fat was a certain indication of its inability to digest it. The sequence of the development of these digestive functions was, therefore, first proteid, then carbohydrates, and lastly fat. It was absolutely in the reverse order that these functions disappeared in dyspepsia. The first function to go in dyspepsia was the digestion of fat. The dyspeptic recognised unconsciously that he could not digest fat, and he avoided it. Again, all the other functions of digestion might be perfect, but the digestion of fat might be imperfect. In illustration of this he instanced the case of a patient who consulted him for indigestion, and improved under treatment. One night he dined out, and was served *inter alia* with spinach, which he ate with relish and digested with facility. So pleased was he that the next night he ordered his own cook to serve him with spinach, but this brought back all his former symptoms in an acute form. The explanation was simple. The first night the spinach had been prepared by a French cook, who had cooked it *au jus*—that is to say, in a solution of meat albumen, whereas on the second

occasion the English cook had cooked it in butter, which he could not digest. In nearly all cases flatulence and acid dyspepsia were due to mal-digestion of starchy matters, yet almost to the end the digestion of proteid was accomplished. The application of this principle to practice was as follows:—One great principle of practice was to give rest to functions that were disordered. Rectal feeding was out of the question in cases of simple dyspepsia, but that rest could be provided by the so-called Salisbury treatment. They could not approve of the way in which it had been brought before the public, but there was no doubt that it was capable of doing much good, limiting, as it did, the diet to proteid, and cutting off the starches for a time; but other methods were better, and less disagreeable. He remarked that the drawing up of a dietary, even by medical men, was often far from scientific; for the factor of the patient's appetite must be borne in mind, and a diet that might suit him very well to-day might prove nauseating to-morrow. He admitted that it was a puzzle to know what to do with fats, but they must perforce be guided by the appetite of the patient. A man who was bilious hated fats, and desired a lemon squash. Now, the emulsification of fats in the intestines was largely carried out by the fatty acids of the bile, and he suggested that possibly the acids in lemon squash might assist in the process. As regards assistance in the digestion of starches, there were the diastases, which often gave marvellous results. In most cases of dyspepsia one might generally leave the albuminous matters to take care of themselves.

Dr. Allchin, in reply, observed that the last speaker's remarks had an interest, though apart from the subject of the paper. He did not think the suggestion to classify cases of dyspepsia upon a developmental basis would be practicable, and after all, when the proposer came to illustrate his view, it amounted to a classification of the different forms of indigestion—of fat, or starch, or proteid, &c.—such as were dwelt upon in the paper. With regard to the speaker's statement of fact, or alleged fact, as to the order of appearance of the capability of digestion of proteids, starches, and fats, and their disappearance in disease in the reverse order, Dr. Allchin thought a little more evidence thereof would be necessary before the views could be accepted. He would also like to say that the question of fatty digestion was not quite as Dr. Maguire had put it. The weight of evidence went to show that fatty stuffs were actually digested, and were not absorbed in the merely emulsified state. He admitted that this was the view formerly taken, but recent observation was against it. It was at present believed that the greater part of the fats were split up into soaps, &c., and absorbed as such, being reconverted into neutral fats during their passage through the cells of the intestinal mucosa, and in the tissues intervening between the surface of the mucous membrane and the lymph channels. It followed, therefore, that the digestion of fats was quite as complicated as some of the other alimentary principles. As to whether the ability to digest fat was really lost by dyspeptics before that of digesting starch and proteid, he was scarcely able to accept that view, at any rate in the form in which it was put, because there were many diseases of the stomach in which the interference with the digestion of proteid occurred from the first. Moreover, the statement that the power to digest fat was not fully developed until after puberty was scarcely consistent with the general facility with which milk is digested by the infant and child, and the readiness with which artificially-added fats, such as cod-liver oil, are absorbed, and therefore digested, by the young.

October 24th, 1898.

FOUR CASES OF ABDOMINAL SECTION FOR SEVERE INJURIES WITHOUT EXTERNAL WOUND.

By A. Marmaduke Sheild, M.B., F.R.C.S.

It may truly be said that in few departments of surgery have there been greater revolutions in practice than in the treatment of abdominal injuries. Wounds of the abdomen are now seldom treated on the speculative plan, and the modern surgeon in such cases does not hesitate to explore the seat of injury, to examine for himself with his own eyes as to whether the peritoneal cavity be really opened or not. By this means lesions of the intestines or other viscera are detected and repaired, and septic wounds of the peritoneum may be cleansed and their baneful results prevented or rendered comparatively harmless.

It is otherwise with that large class of injuries in which no external wound occurs. Here, even at the present day, the rules of practice are by no means clear. Accordingly the experience I shall bring forward to-night may not be without value, for it is only by the systematic publication of such cases that real knowledge increases and guiding principles of practice are ultimately established. I may generalise and say that there are few modern surgeons who would treat a severe abdominal injury now on the so-called expectant plan, *i.e.*, giving opium and leaving the case to take its chance. At the same time, we have to be exceedingly cautious to avoid running into the opposite extreme and doing needless operations.

These cases are common enough in country practice and concern every one of us, and the subject is one which must be of especial interest to those practising in remote districts.

Now, it may first be pointed out that when a patient is subjected to severe crushing violence, such as the passage of a heavy wheel over the abdomen, the symptoms of rupture of certain viscera are generally clear enough. Especially is this the case in injuries of the kidneys or bladder. The signs of rupture of these organs are well enough known, and the correct surgery of ruptured bladder and ruptured kidney has been sufficiently exemplified

by many published cases. What remain, therefore, are injuries of the stomach, intestine, liver, spleen, pancreas, mesentery, and great nerves, and it is in these that the greatest difficulties arise in diagnosis and treatment. The symptoms, especially if masked by drunkenness, are often very obscure, according to the degree or extent of the injury received. Most patients from fright and pain are in a severe state of collapse after an abdominal injury, but this collapse may pass off and improve after a few hours. I should look upon the persistence of collapse as being a grave sign of internal injury. Vomiting and rigidity, with subsequent distension of the belly and increasing quickness of the pulse, are also symptoms of serious omen. Localised acute tenderness and pain must not be ignored. But there will yet be plenty of cases which present themselves where doubt will occur. I can only say that in my own practice I look upon a small median opening into the abdomen, if quickly done, as being very free from risk, and this at once tells us if there be intra-peritoneal extravasation of blood, escape of gas or fæces, or stomach contents. If such be found, immediate enlargement of the wound may enable us to deal with these conditions.

With these preliminary remarks, I now proceed to relate four cases which, I venture to think, are of much interest, and I shall conclude by drawing lessons from each of them.

The first case affords a striking instance of the value of abdominal exploration in injuries where signs of internal lesion are present without external wound.

A boy, aged 11, was admitted into St. George's Hospital on July 8th, 1896. He had been run over by a cab; the wheel passed over the abdomen just below the umbilicus. He was very collapsed, and complained of severe abdominal pain. I saw him on July 9th. He lay in bed with the knees drawn up, and moaned and cried if the abdomen was touched. The face was anxious and pinched, and there were dark circles under the eyes. The temperature was 101°, the pulse 138, and very small. He had vomited twice, but had passed water naturally. Clear urine had been drawn off by the catheter.

I opened the abdomen in the median line, and found it greatly distended with dark clotted blood, like a case of ruptured extra-peritoneal gestation in the female. This was sponged away, and a source of smart bleeding was found in a considerable tear of the

mesentery near its root. On rubbing with a sponge hæmorrhage occurred, and a large vein was tied with catgut.

The rent was sewn together, and the abdomen washed out with warm water. The liver, spleen, and intestine were apparently free from injury. The boy made a rapid recovery, the after history being quite uneventful. He was strikingly relieved of pain by the operation.

The next case had a more unfavourable termination. On the night of April 14th, 1897, I was called to St. George's Hospital to see a boy, aged 14, who three hours before had been run over by a heavy dray. The right thigh was fractured in the middle third also, but the pelvis seemed intact. The urine was drawn off by a catheter, and was clear. The boy had vomited, he was anxious and collapsed, and the belly was rigid and exquisitely tender. The pulse was quick and weak. After a few hours of warmth and rest the pulse improved, and I opened the abdomen. It was full of blood, which welled up copiously. I at once passed my hand to the spleen and liver, which felt intact. Hot water flushing removed the blood and clots, and a large tear was found in the mesentery severing some large vessels, which were soon secured. The bleeding here was very profuse, and I could not doubt but that some large artery had been injured. The jejunum opposite the mesenteric injury was torn right across. Pieces of elastic tubing were placed upon the jejunum on either side, and the two ends cut off with scissors. End-to-end anastomosis was then done by a double row of silk sutures. The wound was joined, but iodoform gauze was packed round the sutured gut and brought out below, for the mesentery was so injured that I feared for the safety of the intestinal junction. The boy was very collapsed, but rallied under saline injections. He had again a relapse, and died in about eight hours. At the autopsy abundant lymph was found round the repaired intestine, and it is worthy of note that it held water, though injected with considerable pressure. A very important discovery was, however, made: there was a second rupture of the intestine, found near the transverse duodenum. This was not complete, but had allowed of the escape of bile and intestinal contents. The second lesion had been quite overlooked at the operation. Had it been discovered I doubt if the condition of the patient would have permitted of its repair.

The next case illustrates an abdominal section for a very desperate condition—rupture of the liver—and is mainly of interest in showing how hæmorrhage may be arrested even from a large rent of that organ.

A boy, aged 16, was admitted into St. George's Hospital on September 7th, 1897, having been run over by a heavy van, the wheels of which had passed across the upper part of his abdomen. He showed all the signs of very grave abdominal injury, and was so collapsed that he seemed likely to die, and any operation was out of the question. I ordered warm injections of strychnia, and copious enemata containing very small quantities of brandy. He rallied under this treatment, but vomited frequently, and complained of agonising pain about the upper abdomen. The belly was now swollen, and its walls were rigid. No blood had been found in the urine or vomit. I was uncertain as to the diagnosis, but inclined towards rupture of the liver. I proposed to open the abdomen on the morning of the 8th, but this was most unfortunately refused by the relatives.

In the evening of the same day the boy was almost comatose, and his pulse could hardly be counted at the wrist. The friends were now anxious that anything should be done which afforded even faint hope, and I accordingly operated. A quantity of extravasated blood and clot was found in the abdominal cavity. This was flushed away with hot water, when the source of hæmorrhage was apparent, from blood welling up in large quantities from the region of the liver. I at once passed my hand to the upper convex surface of that organ, and here I found a large rent, the hepatic substance being quite smashed. I instantly stuffed this with a large sponge wrung out in turpentine, while many yards of strips of iodoform gauze were being prepared. The sponge was removed and the gauze substituted, being packed firmly in with the fingers. I had the satisfaction of seeing that this completely controlled the bleeding. A counter opening was rapidly made below the right ribs, and the ends of the gauze brought out through it. The abdomen was then flushed and cleansed, and the wound closed. It was obvious at the close of the operation that the case was hopeless, or nearly so. The boy survived only eight hours, and it seemed as though the operation had been undertaken too late. At the autopsy I found many large hepatic veins firmly filled with clot, and the appearance made me think and believe

that the hæmorrhage from a ruptured liver might, in less severe cases, be successfully dealt with in the way I have narrated.

The next case illustrates forcibly the inherent difficulty attendant on the diagnosis of these injuries.

On the night of May 6th, 1898, I saw a woman, aged 61, who had been run over by a cab. The wheel had passed over the lower abdomen. She was very collapsed and showed all the signs of grave abdominal injury. The catheter had drawn off some urine slightly tinged with blood. The bladder, however, held 8 ounces of injected boracic lotion, and from that circumstance I was of opinion that this viscus was not ruptured. I thought it better to explore the abdomen. This was done, and a little bloody serum was found in the pelvis. There was an ecchymosis on the posterior surface of the bladder about the size of a shilling just where rupture generally occurs, and I imagined that the symptoms were due to this. No lesion could otherwise be found.

The case did not do well, and it soon became obvious that I had overlooked some injury of importance, though it was difficult to surmise in what direction the oversight had occurred. The abdomen became distended, vomiting was urgent, and febrile symptoms supervened. On the third day she became violently maniacal and tore out some of the sutures from the wound. The bowels were freely opened on the second day by saline purges, with no benefit. She died on the fourth day, exhausted by maniacal delirium and fever. The autopsy revealed the truth. A quantity of extravasated fæcal matter was found behind the cæcum, with diffuse suppuration extending up the retro-cæcal tissue towards the kidney. The rent in the cæcum was small and covered with lymph, and it could have been repaired with ease during life, had it only been discovered. The lesion was not detected at the autopsy until the pathologist had removed the greater part of the intestines.

These cases suggest many points of interest and importance. The first is an instance of intra-peritoneal hæmorrhage from torn mesenteric vessels, and it illustrates how easily this condition may be dealt with. Though it is true that extravasations of blood within the peritoneal cavity may be encysted or ultimately absorbed, no one would now leave such cases alone if the bleeding, as in this case, be excessive and sufficient to

distend the cavity of the abdomen. Excessive peritoneal hæmorrhage after injury is unfortunately too often associated with ruptures of some large viscera, especially the spleen and liver, and it is to these organs that the surgeon should first direct his examination where large collections of blood present themselves on making the opening.

The second case further illustrates this point, for the bleeding was associated with complete rupture of intestine. This serious lesion was readily repaired, yet a second and obscure rupture was overlooked. Nothing could more strongly show us the difficulties the surgeon has to contend with in these cases. The injuries are often multiple, and finding one and dealing with it occupies the mind and attention of the surgeon to the exclusion of others. The last case related is a peculiarly striking instance of this. The blood in the urine and the ecchymosed bladder seemed sufficient explanation of the symptoms, yet a lesion was found at the back of the cæcum which caused death, but which could have been readily and rapidly repaired. I fear we shall never certainly be able to find such injuries as these during life. The localising symptoms are very vague, and the distended intestine prevent an accurate search, unless, indeed, we are prepared to actually eviscerate the patient, as in the *post-mortem* room. The only practical lesson of importance we can draw from these two cases is to recollect that our search must be very thorough and methodical, and we must not allow the discovery of one injury to negative the possibility of another also existing.

As regards the case of rupture of the liver the conditions were obviously hopeless, but I have related the case because it raises the question of the best method of dealing with these very desperate injuries. I was much impressed with the value of the iodoform gauze tampon in this case, and had operation been performed earlier, before the patient had well nigh died from hæmorrhage, the result might have been very different.

In conclusion, it may be pointed out that the mortality of this class of operations must always be high; it is not the operation which kills, but the too often desperate conditions for which it is undertaken. Unsuccessful operations must be looked upon as merely failures to save life, not as fatal in themselves.

Their high mortality must not deter us from operating. Occasionally we shall save a life otherwise doomed to speedy and certain destruction.

Dr. KINGSTON FOWLER (Vice-President, in the chair) said that he was interested in the fact that in several of the cases two lesions were discovered. It was well known that in cases of chronic obstruction the obstruction might exist in more sites than one. He asked whether in the cases of rupture of the intestine there was obliteration of liver dulness, as often occurred after perforation of a gastric ulcer. He mentioned that some years ago he had believed that such cases inevitably terminated fatally unless operated upon. An out-patient under his care for chronic gastric ulcer became the butler to a friend, and one day he was asked to see him. The man had suddenly become collapsed and it was evident that perforation of the ulcer had occurred, although the liver dulness was normal. On the following morning there was complete obliteration of the liver dulness. The patient refused operation. The resonance over the liver gradually disappeared, and eventually the area of liver dulness became normal. Recovery was complete.

Mr. G. R. TURNER said the paper was the more interesting to him, inasmuch as he had seen two of the author's cases. The first case he himself had operated on was in 1892, and had been brought before the Society. It was that of a small boy who had fallen 12 feet on to the pole of a boat, and then into the river. On admission he was extensively collapsed, and vomited. Twenty-four hours later Mr. Turner opened the abdomen and found an extensive twist of the ileum, in fact the boy had traumatic volvulus ; this was released and he made a good recovery. Since then he had had at least four cases in which he had opened the abdomen for injury, without external wound. His next case was one of rupture of the liver, in which there had been extensive bleeding into the abdominal cavity. He found a rupture on the under surface of the organ, which he plugged with gauze. The patient, however, was much collapsed, and soon died. Mr. Turner also related an hitherto unpublished case in which a man had received a blow on the abdomen causing rupture of the small intestine. There was much collapse and vomiting. On opening the abdomen he stitched up the rupture and, at the same time, did the radical cure of a hernia. The man made a perfect recovery. In another case the collapse on admission was so extreme that he had to delay operating. On opening the abdomen he found a little free blood, but no hæmorrhage was going on at the time. There was a slight rupture on the under surface of the right lobe of the liver, which was in process of cure. The whole of the retro-peritoneal space was filled with an enormous extravasation of blood which extended along and surrounded the large vessels at the back of the abdomen, which was firm and hard to the feel. He diagnosed ruptured kidney but thought it better not to interfere, especially as it did not appear to be extending. The patient recovered from the exploratory operation, but vomited persistently until he died some 10 days after. *Post-mortem* there was no peritonitis, and only a slight injury to the under surface of the liver, which was convalescing, and there was nothing to account for death except the enormous hæmorrhage from the badly crushed kidney. The following week another patient was admitted with almost identical symptoms—collapse, vomiting, and blood in the urine. The patient was a man who had fallen from his cab, striking his loin against the kerbstone. When he recovered from his collapse it could not be ascertained with certainty whether any other organ was wounded, so an incision was made through the linea semilunaris. It was found that there was nothing wrong with the general peritoneal cavity, but there was exactly the same condition of the retro-peritoneal

space as in the previous case, though in an earlier stage, and rapidly extending dulness was noted in the right flank. On opening the peritoneum to the outer side of the colon Mr. Turner found that the kidney was torn right across. It was impossible to distinguish blood clot from kidney, and eventually he removed the two halves of the kidney. In spite of bronchitis, the man made a good recovery. Out of these five cases, one of ruptured gut, one of ruptured kidney, and one of traumatic volvulus of the small intestine had recovered. Mr. Turner observed that this was one of a class of cases in which it was extremely difficult for a surgeon imbued with the modern spirit of interference in abdominal injuries to know when to hold his hand. It was true that in the olden days many cases of obscure abdominal injury recovered under treatment by the expectant method, but others figured in museums. The surgeon was always exposed to the risk that the shock of operation might prove to be the last straw, so to speak. In presence of a patient, collapsed after severe abdominal injury, the modern surgeon was impelled to open and see what it was. In general, no doubt, that was right ; but in a certain proportion of cases the patient would recover even if the abdomen were not opened.

Dr. F. Lucas Benham referred to a case of presumed rupture of internal viscera which got well under energetic medical treatment. The patient was a lad who slipped while running in the street, his side striking the kerb. He complained of much pain and tenderness just over the cæcum. Shortly afterwards peritonitis supervened and soon became extensive, as well as severe, the abdomen becoming greatly distended and tympanitic, with vomiting and intestinal obstruction. Dr. Benham put on leeches and gave opium, with mercury both internally and externally, and when dangerous symptoms appeared of obstruction with stercoraceous vomiting, he used turpentine to relieve it, and these measures were followed by recovery.

Mr. Wallis said he was glad to hear the author lay stress on the harmlessness of a properly-conducted abdominal section. Some of the cases he had mentioned were specially interesting, notably the two in which the mesentery had been wounded. He was much interested in the first case by reason of the fact that in spite of the extensive injury to the mesentery no gangrene of the intestine had followed. He recalled a paper that he had brought last year before another society, in writing which he had had to look up a large number of cases of injury to the abdomen from various causes. In a large proportion of the cases recovery had ensued without operative assistance, but a fair percentage of the cases became septic, suppurated, and died ; although, as was subsequently shown in the *post-mortem* room, some of them might have been saved by early operation. Of course it could not be settled in a hurry when to interfere and when not to interfere. One point not touched upon by the author was the extreme injury done in some of these cases to the solar plexus and to the nerves which governed the abdomen, and the shock which these injuries would entail could not but be serious. It often took hours and even days for patients to rally. He recalled a case, reported from Germany, of a man who was stabbed soon after Carnot was assassinated. The liver was wounded, but assistance being near at hand, the abdomen was opened and the wound dealt with by means of sutures, and an excellent recovery was made.

Mr. Battle observed that these cases of abdominal injury unattended by wound of the abdominal wall gave surgeons a great deal of anxiety

He had long since given directions to his house surgeon that all such cases should be closely watched from hour to hour, and the diagnosis made as soon as possible, so that surgical treatment might be resorted to if indicated. It was difficult to compress within short compass the difficulties which were met with in the diagnosis of these cases; as, for instance, the question of dulness in the abdomen as indicating the presence of fluid, which was perhaps the most important one, especially in cases in which there was no rupture of the bladder. If the patient's condition was getting worse, with increasing fluid in the abdomen, pointing to the probability of extravasation of blood still going on, he had directed that he should always be sent for. It was not easy to diagnose one group of cases—that which simulated rupture of intestine. He himself had operated in three cases of rupture of intestine. In the first case the symptoms were very severe indeed—vomiting, pain, collapse, and great rigidity of the abdominal muscles. In the second case, operated on this year, peritonitis had already set in, it being three or four days after the injury. The patient's general condition, however, was quite calm, the mind being quite clear, the pulse only 90, and the temperature about normal. There was distended and tender abdomen which was rather tense; he operated and found ruptured intestine. The third case had been sent away from another hospital, he having been drunk at the time of the accident, and there being no signs pointing to abdominal injury when examined. The accident occurred about 6 p.m., and he saw the patient on the following day at 2 p.m. He was vomiting with rigid abdominal walls. He operated, but the man died a month later of subdiaphragmatic abscess which he had been unable to find. These were cases in which one was justified in suspecting ruptured intestine, and in such cases he thought it was right to operate. French surgeons had laid it down as an axiom that if these cases were not operated on within 15 hours they could not be saved. He alluded to the case of a man who was run over and sustained an injury of the abdomen, though not of a very marked character. He lay quiet in bed with a normal temperature until the fourth day, when his temperature ran up to 102° or 103°, and he died with rapid collapse. In that case there was a small rupture of the intestine. Then as to the question of shock. That was a very variable symptom, and one of the most unsatisfactory, because in comparatively severe injuries there might be no shock, while it was often well marked in comparatively slight cases. He remembered a case of ruptured liver at the posterior part in which, on admission, there were no abdominal symptoms and no collapse, yet in three hours the abdomen was full of blood and they were unable to stop the hæmorrhage. One never knew in an abdominal case how soon unfavourable symptoms might supervene. He suggested that Mr. Benham's case might have been one of typhlitis associated with some peritonitis. He asked the author what was the condition in his cases with regard to dulness on percussion of the abdomen.

Mr. Turner said he had seen the man about three hours after the accident, and had told him that in all probability he had ruptured intestine, but the patient refused operation. It was not until some eight hours later that he consented.

Mr. Sheild, in reply, said he would be sorry for it to go forth that a surgeon ought to open the abdomen in all grave cases of abdominal injury. It was only when the collapse did not pass off, and the belly became rigid and distended, that operation was urgently called for.

Persistence and progression of bad symptoms indicated exploration. He said the case related by Dr. Benham was an excellent example of the opposite kind. In that case the boy recovered, but one could not be sure in any given case what was going to happen. Appendicitis could be provoked by injury, as was well known, and Dr. Benham's case could probably thus be explained. By way of contrast, he recalled a case which he had seen last year, the patient being a woman who had been run over and had bruised the right side of the abdomen. There was much pain and fever, and every sign of grave abdominal injury. He urged an exploratory operation over the cæcum, but this was not done, and she was treated by the application of leeches, and made what was thought to be an excellent recovery. Ultimately, however, a large abscess formed in the loin with fæcal fistulæ, from which she died. There was probably a rupture in the neighbourhood of the cæcum. He said that if one were to ligature the mesentery extensively or to tie a large artery, gangrene of the gut would probably result. But the mesentery bears extensive injury without gangrene necessarily taking place. He was glad attention had been called to the difficulty of diagnosing some of these cases. These were the cases that occasionally gave rise to so much trouble before coroners, and over which many a medical reputation had been wrecked. He urged that a slight injury to the abdomen should be regarded and treated like a slight injury to the head, that is to say, with the possibility clearly before them of more serious symptoms supervening at short notice. In this class of cases he had had great difficulty in obtaining dulness on percussion when there was undoubtedly extravasation of fluid. The intestines rose on the surface of the fluid and masked the dulness. Another important fact was that if the patients were suffering from pain and tenderness they would not stand much percussion and tapping. Again, the disappearance of liver dulness was a symptom which should be looked upon with great caution. If well marked, it was a symptom of great value, but he had seen it simulated very closely by distended intestine, and he had seen it diagnosed when it did not exist. No strict rules could be laid down for the guidance of surgeons, and each case must be treated on its merits. On the whole, in spite of the fact that a certain proportion might recover without operation, he thought that most of them would stand a better chance from operative treatment than if treated on the expectant plan.

ON THE ADVANTAGE OF VENESECTION IN ACUTE PNEUMONIA, WITH CASES IN ILLUSTRATION.

By F. Lucas Benham, M.D.

I think that it may be said with truth that the prejudice against venesection has diminished of late, and that the reaction against the old-fashioned excess of the practice is not as strong now as it was some years ago. For all that, blood-letting has by no means regained as yet a firm hold in the medical profession,

and its employment is rare, and never extensive. It is even still loudly condemned by many authors and others whose voices are most heard; but I fancy that the most emphatic denunciations proceed from those who have had no experience whatever of the practice, and who are either biassed against it on theoretical grounds, or are perhaps too timid to resort to it. I have sometimes heard medical men say that bleeding seems reasonable enough; but suppose the patient dies in spite of it, his friends will very likely ascribe the fatal result to the bleeding, and cause unpleasantness. In answer to this, the advantages of bleeding are usually so evident that this difficulty is not likely to arise.

Still, utterances are heard in its favour from time to time, either in connection with some reported cases where it has been successfully but casually employed, or as a sort of pious opinion, expressed in some such way as this :—"Though it is unquestionable that this method was carried to gross excess in former times, yet we may now, perhaps, have drifted too far towards the opposite extreme, and some good might be obtained by resorting to it occasionally to a moderate extent."

It is not my intention to discuss on the present occasion the subject of venesection in general and all its applications, but to confine myself to its use in acute pneumonia. I limit myself thus for these reasons :—(1) The whole subject is too large for a single short paper. (2) Pneumonia is a common and familiar disease, and affords everyone opportunities of testing the method I recommend; and, besides, it is *par excellence* the disease in which its advantages are evident. (3) I have had more experience of bleeding in this disease than in any other, or group of others, and as even the smallest contribution may be of value to guide and assist others in adopting any course of treatment, I hope that the following set of cases may be beneficially recorded.

Before going further, I wish to say that I have only employed blood-letting (and by blood-letting I mean almost always bleeding from a vein at the bend of the arm) in very severe cases of pneumonia—*i.e.*, cases that were rapidly getting worse and developing dangerous symptoms, and seemed to have small chance of recovery, or which were specially bad in one way or other, so as to require special relief; and I have taken no account of mild

or average cases which recover spontaneously or under ordinary treatment. Nor have I made a complete series of all cases that I have bled, so as to include all those which died in spite of bleeding; for, of course, I do not claim to have saved every case. Statistics are very fallacious. But I can confidently say that cases that have ended fatally after bleeding have almost always been those where it was not resorted to till too late, or perhaps insufficiently, and I am sure that in no case at all has bleeding done any harm whatever.

I may also, to show the converse side of the question, refer to one or two cases that died without having been bled, which, to my regret, probably slipped through my fingers in consequence of my taking too favourable a view of the prognosis, and died contrarily to my hopes and expectations; whereas, if I had bled them in time, I might very likely have saved them.

The cases I have to relate, the notes of which I have epitomised as much as possible, are as follows :—

Case 1.—The first case of acute pneumonia I ever bled was that of A. S., male, aged 22, a bottle washer, who was supposed to have caught the disease from exposure to the effluvia of drains. He was admitted to the St. Marylebone Infirmary on July 13th, 1882, on the fourth day of his illness. The temperature then was 104° F. On the nights of the 14th and 15th he was wildly delirious, rushing out into the passage, breaking windows, and resisting and fighting actively. In the afternoon of the 16th (seventh day of disease) the temperature had fallen to between 101° and 102°, but his condition was extremely grave owing to prostration following the excitement, and acute distension of the right side of the heart. He was almost unconscious from stupor, his face of a livid or leaden hue, the pulse 160 and rising, and respiration very rapid and shallow. In fact, he was almost moribund. As a last resource, and prompted by the recorded acts of the old authors, I bled him to 10 ounces. His condition thereupon gradually improved; in the evening his pulse was down to 126, and he had a quieter night. Next day (July 17th) the temperature was 103°–104°, the pulse 120–136, and he was again bled to 9 ounces. After this he improved greatly, slept, and appeared to be out of danger. The crisis approached, the temperature falling till it reached the normal on the 19th (the tenth day of the disease); it remained subnormal, and he was soon perfectly convalescent.

But for the timely bleedings, he certainly would not have survived till the crisis. Better and more quickly decisive results might possibly have been obtained by drawing blood more freely in the first instance.

Case 2.—T. P., male, aged 18. Admitted to the St. Marylebone Infirmary on August 7th, 1883, with a severe attack of acute pneumonia; his chances of recovery seemed very doubtful, and he was in a good deal of distress. The temperature rose to between 103° and 104°, and the pulse on the evening of the 8th was 128 the tongue was very dry and

coated. V.S. 10 ounces. In 24 hours the temperature had fallen below normal, and he became convalescent. The bleeding here tided him safely over the crisis, and gave him some relief.

Case 3.—M. M., female, aged 44, the subject of chronic phthisis, for which she had been in the St. Marylebone Infirmary 12 months before, and had been discharged, relieved. She was again admitted on January 1st, 1884, with acute pneumonia on the left side, with which she had been ill for six days. The patient was ill-nourished, thin and weak, with much dyspnœa and a good deal of cyanosis; the temperature was 101°–102°, the pulse 130. Prognosis seemed very bad. She was bled to 10 ounces late in the night after her admission; the clot was soft and red, not buffed or cupped. She seemed much the better for the bleeding. In two days the temperature fell to normal, though there was a slight subsequent rise for a few days, and she recovered afterwards. This case is of interest, as it shows that blood-letting may be safely and beneficially employed in phthisical patients. It has been asserted further, by those who have tried it, that no remedy is so effectual in controlling phthisis in its early stages as small and repeated bleedings, but I have had no experience of it, except in the form of spontaneous hæmoptysis.

Case 4.—B. S., female, aged 18, admitted to the St. Marylebone Infirmary on December 7th, 1883, with acute pneumonia of five days' standing, the whole upper lobe of the right lung being affected. The temperature on admission was 102°–103°, the pulse 108, the tongue moist and furred. Very sleepless. Next day the tongue was dry and brown, and the pulse had risen to 126. V.S. 10 ounces; later in the day the pulse had fallen to 100. Next day the temperature had fallen to normal, and the patient recovered.

Bleeding was performed in this case because of the rapidity of the pulse, and the patient's general condition seemed to be rapidly becoming worse, and it was not certain how long it would be before the crisis took place. If life was not actually in danger, yet the bleeding gave great relief, and did no harm.

Case 5.—Acute Bright's disease with acute pneumonia. L. K., male, between 30 and 40 years of age, of medium height and rather stout, had suffered a good deal from rheumatic gout (by which I mean a form of genuine gout), for which he had been admitted to the St. Marylebone Infirmary on two previous occasions, and he had had no albuminuria then. He was admitted again on April 11th, 1883; he came from a convalescent institution, on account of acute eczema of the forearms and legs, produced by the application of petroleum, which he had been rubbing into his joints; and he had increased the effects of this by sitting before the fire until he nearly set himself alight.

After his admission his urine was very scanty; he said it had been almost suppressed for several days after using the petroleum; on the 18th only 9 ounces were passed; it contained albumen, and deposited numerous fine hyaline and some epithelial casts; by the 24th it had increased to 55 ounces per diem, and was of low specific gravity. There was some œdema of the legs, which remained after the eczema had subsided.

Soon after his admission bronchitis developed, with dyspnœa, severe nocturnal asthma, and difficult expectoration; later pneumonia came on,

a patch of distinct consolidation over the left scapula, with rusty expectoration, &c. On April 23rd he was becoming much worse. In the afternoon of the 25th he had urgent dyspnœa, became cold and collapsed, and appeared moribund. Pulse was imperceptible. V.S. 9 ounces at once. Two 1-drachm doses of Vin. Ipecac. were given, and he gradually improved; and still further after the application of numerous wet cups, followed by turpentine stupes to the back. The temperature, which had been 102°, gradually fell, and reached normal on the 29th. He recovered entirely.

Case 6.—C. S., female, aged about 30, admitted to the St. Marylebone Infirmary on October 8th, 1882, with acute pneumonia; the temperature was then 101°–102°, but rose early next morning to 105°, and at 3 p.m. was 103·4°, the pulse 140, and respiration 60. V.S. 12 ounces. The following morning (October 10th), temperature 103°, pulse 112, respiration 58. At 3 p.m., temperature 103·5, pulse 150, respiration 70. V.S. repeated, 9 ounces. The patient was easier after this, though the temperature remained above 103°, and the pulse between 130 and 140. On the 12th (two days after the second bleeding) the pyrexia began to abate, and the temperature became normal next day. The patient recovered completely. This was a severe case, and was, I think, greatly mitigated by bleeding. It is uncommon for cases of pneumonia in adults with a pulse of 140 or 150 to recover. Possibly the continuous application of cold compresses might have been equally effectual. I employed the latter method with success in some cases, *e.g.*, on a lad, aged 16, with acute double pneumonia, temperature 105, and pulse 144, and in the following.

Case 7.—J. R., female, aged about 60, admitted to the Infirmary on April 9th, 1883, with acute pneumonia in a severe form on the right side; the temperature was not very high, seldom exceeding 103°, but the pulse rose to 120 or 130, and the patient was very ill. V.S. 7 ounces on the 10th, and again to 9 ounces on the following day; the pulse having become very rapid, and there being much dyspnœa. The temperature became normal on the 12th.

In this case cold compresses were applied to the chest from the first, which no doubt contributed to moderate the symptoms; and I noticed that on every fresh application of the cold compresses the dry brown tongue became moist and cleaner, the pulse slower, and the patient easier.

Brandy was also given regularly during the progress of the case, but I am not at all sure that this did any good and no harm.

Although the patient was practically convalescent from the pneumonia, diarrhœa came on two days later, and lasted several days; and finally an attack of apoplexy (probably from cerebral hæmorrhage) with right hemiplegia seized her on the 25th, and carried her off in 24 hours, the temperature *post mortem* being 109·2°.

Case 8.—Mrs. M., aged 48. Rigor on March 5th, 1887. On March 6th, temperature 104°, pulse 108–120; temperature remained the same. The illness proved to be a severe attack of pneumonia, the base of the left lung being affected. On the seventh day the patient was growing worse, the pulse more rapid. There was prostration and also slight delirium. V.S. 10 ounces. After this the patient was easier and better, though

the pulse remained about 130 until the crisis; the temperature fell gradually, then suddenly, reaching normal on the 14th. It immediately rose again to 103°, and kept more or less irregularly raised till April 9th; but though the illness was protracted and severe there was no danger in the latter period as there was in the first. Patient eventually recovered and has been a healthy woman since. There was albuminuria in this case during the attack.

Case 9.—Mrs. H., aged 47. Rigor on November 7th, 1893. On November 9th there were pain and crepitation in the upper part of the left side of the chest, and hard, frequent cough, with glairy rusty sputa. Two leeches gave relief from the pain. Temperature was highest, 103·6°, on the 11th. Leeches were again applied on the 13th. On the 14th, though the temperature was beginning to fall, the pulse and respiration were becoming more rapid, and the patient was sleepless and delirious at night, and prostrate, dull, and partially delirious by day. On the 15th, at 5 p.m., the pulse was very rapid, small, uneven, and occasionally intermittent. V.S. 13 ounces. Patient slept well afterwards, and was much better next day. The attack was followed by rather persistent diarrhœa, perhaps partly due to calomel having been administered somewhat freely, as I believe that bleeding may be less urgently required if calomel is steadily given. Patient quite recovered, but died two or three years later of cancer of the uterus, which ran a rapid course.

Case 10.—Mrs. S., aged 72, a healthy woman, who did not look as old as this. On November 22nd, 1893, the weather being very cold, she was seized with violent retching and sickness in the morning, which lasted all day. I saw her next day, and found her suffering with fever, headache, and cough. On the 25th there were pain in the left side and physical signs of pneumonia of the whole left lower lobe. Two leeches were applied and bled well. Next day the pain was gone. Pulse 120, respiration 26, temperature 103°. On November 27th patient was very weak and had slept but little. Temperature 100, pulse 118, sputa rusty. V.S. 8 ounces, clot buffed and cupped. On November 29th physical signs had cleared up over the base of the lung, but were still heard over the scapula. The pulse was still rapid, 134, and the tongue was very dirty, and the patient was prostrate, heavy, and sleepy. Next day the temperature was normal. Convalescence was delayed by a very bad aphthous sore throat, which appeared during the attack of pneumonia; at first I almost suspected the aphthous deposit to be diphtheritic membrane. It was slow in disappearing, and the patient eventually recovered completely, and is still well and active. This case shows that it is quite safe to bleed a patient over 70 years of age, and that advantage may be derived from it.

Case 11.—Mr. S., aged 57, carman, a healthy, fairly robust man, who had had an attack of gout twelve months before. While driving his cart on October 26th, 1887, his horse fell, and in trying to raise the animal, with his right arm under the shaft, he thought he must have strained his right arm and shoulder. He had a rigor and vomited that night and again next day, but went to work. On the 28th he gave up work, felt very sick, and went to bed. Temperature 102·3°, dry cough, much pain in the right side. No fracture of the ribs or any other injury was to be

found. Pleuritic friction and crepitation were heard, and three leeches were applied to relieve the pain.

The attack of pneumonia was of moderate severity, but the man was tormented with utter sleeplessness, and complained constantly of it. I told him that blood-letting was the only sure remedy for this, and he willingly consented to have this done, as he had formerly lived in the country and had often seen people bled in his younger days; so on October 30th I bled him to about 12 ounces, the clot being much buffed and cupped. The following night he slept better, and had no further trouble of the sort. Temperature began to fall on November 1st, and reached normal on the 4th. A week later, an attack of gout followed, as it usually does after acute diseases, where the predisposition exists.

Case 12.—A traumatic case: double pneumonia from fractured ribs, caused by a violent injury. J. L., aged 64, coachman, whom I had attended over and over again for attacks of gout and bronchitis, and once for severe influenza with pneumonia. He was a spare man, of medium height, temperate, and in good health at the time. On June 6th, 1894, while driving a carriage late one evening the horse ran away, and the man was thrown off the box into the road; he was only stunned for a short time, if at all, and received a slight scalp wound. He was taken home and seen by me at 11.30 p.m. He complained of pain all down the left side, from the shoulder to the lumbar region; the sixth and seventh ribs and perhaps more were broken on the left side, but I did not detect any fracture on the right side. The back was extensively bruised. The slightest movement caused most intense pain. The ribs were strapped and bandaged. There was no hæmoptysis. He slept badly and had more or less constant pain and distress for the next few days. On June 10th he complained of severe constrictive pain round the chest. Nothing was found on physical examination, and the temperature was normal.

Next day he was very much worse, with a heavy, anxious, and distressed expression; breathing rapid and embarrassed, expectoration sticky and slightly rusty; temperature 102°, pulse 108; tongue foul. Physical signs of pneumonia were found over the left lung and the base of the right lung. At 9 p.m. he was so ill that I bled him to 20 ounces; the clot was buffed and cupped.

June 12th: He had had a better night. Temperature 100, pulse 96; respiration easier. Pain and distress much less. As he had been so very much benefited by the venesection, I bled him again to 10 ounces. He was very much better the next day; had had a good night, and not much pain was now felt. Temperature 99°, pulse 90; respiration easy; expectoration looser and more purulent. On June 16th he was able to sit up in bed. He speedily became convalescent, and is still alive and well.

Case 13.—E. M., female, aged about 45, lady's maid. Had a severe rigor in March, 1885. Pleuro-pneumonia of the whole of the left lung developed, with rather severe constitutional symptoms, heaviness and somnolence, with pulse and respiration increasing in frequency towards the end of the week. I bled her to about 10 ounces with relief, and again the next day to a less amount. The crisis took place suddenly about 24 hours after this, and her condition then became alarming. I found a loud systolic murmur over the whole of the cardiac area, which must have been due to the formation of a clot on the tricuspid valve. Of

course alcohol was then indicated, and I gave stimulants freely, about half a pint of brandy and champagne also being required daily for nearly a fortnight. The patient, fortunately, was well nursed throughout. She steadily improved and made a perfect recovery. The murmur grew less and less and ultimately entirely disappeared. I saw her from time to time afterwards, and she remained as well as before.

In this case the clot in the heart was one such as is apt to form during the last hours of life about the time of the crisis in acute pneumonia, and is usually the immediate cause of death. I do not think that venesection can bring it about in any way.

Case 14.—Mrs. N., aged 51, very stout, florid, and plethoric; very emphysematous and short of breath. She had suffered for many years from attacks of acute bronchitis, cough, &c., and has kept inside the house constantly for many winters. The heart was somewhat dilated and probably fatty.

On May 8th, 1889, she had been in good health for some time. At mid-day had a severe rigor and went to bed; there was severe pain in the lower part of the chest and over the abdomen on the left side. In the evening I found the temperature 104°, and there was friction and crepitation at the left base behind. Two leeches were applied over the angle of the left scapula.

May 10th: Temperature 103°, pulse 110, regular, but slightly intermittent; respiration 40. Was prostrate and inclined to faint; not delirious.

May 11th: Pulse 110, irregular. I wished to bleed her, but the patient strongly objected thereto, and, moreover, no superficial vein could be seen. Three leeches were therefore ordered and applied to the back and left side. Two of them bit, and one bite continued to bleed till the evening, the blood soaking through the clothes and forming a large pool in the bed, and the patient felt very much relieved.

May 12th: Temperature 99·4°, pulse 100, regular, soft; respiration 30. Pain and distress much less, and the patient had slept much better. Next day—temperature normal, pulse 90.

May 14th: Herpes on left side of the lip. Convalescent.

The woman regained her usual health and survived till February, 1896. During the last year or two of her life she took hardly any exercise. There was considerable albuminuria, dilatation of the heart increased, and there was anasarca. Cerebral hæmorrhage occurred at last, and she died in a few hours. During this attack of apoplexy I made an attempt to bleed her, and after cutting deeply into the bend of the elbow, I managed to find and open a vein at the bottom of the hole, but could not draw much blood, as the opening in the vein closed up. The bleeding was insufficient to make any effect on the apoplexy.

The difficulty of finding a vein in very stout people has led me to wonder how venesection was accomplished in such subjects in bygone days; for assuredly it cannot be effected without a good deal of dissection. Quite lately I attended a woman weighing over 20 stones, a pure case of extreme obesity. To relieve certain symptoms I wished to open a vein, but there was not a single visible vein of any size. I gave temporary relief on two or three occasions by opening a small vein in the front of the wrist, and thus withdrew two or three ounces of blood each time. I postponed doing so on a later occasion, to see if I could not devise a way of bleeding her more effectually; but, unfortunately, in the interval she

died suddenly ; and I have very little doubt that life might have been prolonged in this case by another timely and larger bleeding. I should be much obliged if any Fellow of this Society could advise me how to solve this difficulty.

Case 15.—W. R., male, aged 24, hairdresser. He had had rheumatic fever three times, but the heart was normal, and the man was temperate as far as I knew. The present attack of pneumonia began during the epidemic of influenza, with a rigor on January 10th, 1890 ; he left off work and went home. In the evening the temperature was 104°, and there was friction with sharp pain on the left side. The physical signs were those of slight pleuro-pneumonia on the left side, and there was not much pyrexia, but he was in a peculiar semi-rational condition for the next few days, being somewhat delirious especially at night. On January 14th there did not seem much the matter remaining ; the physical signs were insignificant, and the pulse 80. I was called up to him at 5.30 a.m. on January 15th, and found him actively delirious, thinking people were in the room, and trying to dress and get out. I noticed a remarkable change in the pulse since my last visit ; it was soft and regular then, now it was 120, very full and tense, like a thick hard cord. He was manageable without much difficulty. V.S. 10 ounces. I left him in bed, telling his friends he would probably be quieter in a little time. The delusions did not entirely subside, and about 8 a.m., before the full effects of the venesection had time to be seen, the patient again became restless and tried to get up to go to the police station. His friends took him to the hospital instead, whence he was soon discharged well. I am inclined to think that such cases of pneumonia with disproportionate amount of delirium in sober people is characteristic of one form of influenza. It might have been better to have bled more freely ; but at the time I intended to repeat it if necessary. At any rate, I am glad I did not give narcotics. Morphia, chloral, and all the rest are, I believe, mischievous, and often highly dangerous in such conditions.

Case 16.—Mr. N., aged 43, a waiter, stout and fairly robust, no phthisis. Early in June, 1895, he had an attack of acute pneumonia on the left side, from which he recovered in a few days, and went out, ate well, and had no cough. On June 25th, three weeks after the commencement of the first attack, he was taken ill one night with severe pain on the right side and round the body ; there was no rigor or vomiting. He was very ill and in great distress, and did not sleep. Next day he was still very ill. In the evening, when I first saw him (he was not my own patient, and I saw him in consultation), he was lying crouched up on his right side ; temperature 101°, respiration 44, much dyspnœa, not cyanotic, pulse 120. He was apparently in great danger. There were physical signs of pneumonia over the lower lobe of the right lung ; the upper part of the left side of the chest was hyper-resonant, and the heart could not be felt on the left side at all, but over a considerable area to the right of the sternum. He had evidently some pneumothorax on the left side in addition to lobar pneumonia on the right. V.S. 18 ounces. He was greatly relieved almost immediately, and slept well afterwards. In a week or so the pneumonia on the right side subsided ; pneumothorax remained on the left, and though fluid was suspected, none could be discovered on exploration with a needle. The man ultimately recovered.

Case 17.—Mrs. P., aged 27, a rather tall woman, who had long suffered from emphysema with frequent attacks of bronchitis and asthma. When free from such attacks she was active and healthy. On May 7th, 1897, she caught cold one evening when out in a north wind. She lay in bed next day shivering and very sick. On May 9th, when I saw her for the first time during this illness, she continued to vomit frequently, and severe pain came on in the right side with a dry cough. Two leeches were applied and gave relief. Pneumonia developed in due course and seemed of moderate severity. On the 13th she was very much better in all respects ; could move and talk freely, was cheerful, and seemed to be improving fast. Next day, however, I found her very ill ; she had had a very bad night, and had been delirious, talking, &c., and had had no sleep. The skin was cool, surface rather livid ; pulse 96. She lay on her back prostrate, unable to move, and only half conscious. Tongue dry. V.S. 8 ounces, clot very much buffed. This gave very great relief. The patient was soon out of danger, and speedily became convalescent. The danger in this case must have been due to distension or dilatation of the right ventricle.

Case 18.—G. H., male, aged 33. Had formerly been in the army, and, like other members of his family, had been an habitual heavy drinker. He had been drinking to excess lately. On April 23rd, 1897, he was seized with violent vomiting, and was said to have been unwell (the effects of drink ?) for the last week. On April 25th he began to present restless delirium, like delirium tremens, and acute pneumonia was recognised affecting the lower lobe of the right lung. On the 26th he was very restless and excited, delirious, and troublesome ; he kept getting out of bed and wanting to go to work, and did not at all recognise his surroundings. He looked very ill ; face red and bloated. Cough with thick muco-purulent expectoration, the patient spitting over the bed or anywhere. Breathing irregular and restrained ; no laboured dyspnœa or rapid breathing. Pulse rapid and weak. Tongue dry.

V.S. 15 ounces, performed under some difficulty, as the man was restless. An icebag was applied to his head, though for a long time it was difficult to keep it there. He was quieter a few hours after the bleeding ; the pulse slower, fuller and regular. He was excited and troublesome again at night, but quieter next day, and presently had a very long quiet sleep. After this the symptoms were moderate and he steadily improved. On the 28th, both knees, ankles, and the joints of the hands became painful and swollen (gout ?) which subsided gradually after a few days. Convalescent early in May.

Since then the man has shown symptoms of phthisis, now in an advanced stage. There may have been tubercle present at the time of the pneumonia, I am unable to say ; it was first evident some time later in the larynx, then the left lung, and there has also been diarrhœa—presumably from intestinal tuberculosis. I do not think that tubercle settled first on the seat of the pneumonia. (He died in July, 1898.)

Case 19.—Mrs. B., aged 50, was seized with a rigor on February 5th, 1898, when influenza was prevalent, and took to bed. A severe attack of acute bronchitis set in, with a good deal of cough and dyspnœa. On the 9th and 10th a patch of herpes appeared on the right side of the upper lip, followed in a day or two by physical signs of pneumonia of the upper lobe of the right lung, which afterwards spread to the lower lobe. On

the 12th the patient looked very ill; the complexion was dingy and sallow; prostration; tongue dry, brown, and cracked; expectoration stopped. These symptoms were relieved by copious sweating, and the patient was better next day. On the 14th, however, she was extremely ill, had scarcely slept, and had been delirious through the night; lay prostrate on her back; tongue dry and brown; temperature only 99·6, pulse over 120, respiration 54. V.S. 10 ounces, clot cupped and buffed. Dozed during the afternoon, and was much easier. Next day the temperature was normal, and a fresh patch of herpes appeared on the right *ala nasi*. Early the following morning she became extremely faint for a short time, which probably marked the crisis. After this she steadily improved, and was able to walk about in a few days. She is still well. This is a fairly typical case of influenza-pneumonia, and shows that bleeding is as much called for and may be as satisfactory as in ordinary cases. It has been a matter of observation that when hæmoptysis occurred in pneumonia following influenza, as it often did, such cases generally did better than those where there was none.

In whooping cough there is, of course, a strong tendency to pulmonary congestion and pneumonia, which is always a source of danger, and is not infrequently fatal. In very many of these cases, even in young children, very great relief can be given, and I am sure that I have sometimes seen life saved, by timely bleeding; as the patients are usually small children, not much blood need be drawn, and one or two leeches usually suffice, though the bites may be allowed to bleed. The most convenient site for the leeches is the back, and with due care as to stopping the hæmorrhage after the bites, they are perfectly safe.

For the sake of acknowledging my errors, and to show the danger there may be in abstaining from blood-letting, I will mention one or two cases wherein I regret that it was omitted, and which ended fatally.

Case 1a.—E. C. F., aged 24, who suffered from a moderate amount of emphysema and occasional attacks of bronchitis, so that she always had more or less cough and shortness of breath, and who had had an attack of pneumonia six years before, caught cold in the north-east wind that prevailed about Easter this year (1898) and had a rigor on April 13th. I saw her on the 15th, and suspected pneumonia; and on the next day physical signs of pneumonia in the upper lobe of the left lung appeared. The pulse was very rapid, 120–130, and she complained of insomnia, and there was some dyspnœa and troublesome cough, though not much pyrexia. Next day there was no fresh symptom at all serious, but the pulse continued very rapid, though regular, and she complained much of not being able to sleep. I was much inclined to bleed her there and then, but I thought that there was no immediate danger, and that it might be resorted to with more advantage somewhat later. She died, however, rather suddenly the following night—certainly at a rather early period of the disease. I consider that if I had only abstracted blood

once or, perhaps, twice, I should have relieved and prolonged the case, and perhaps have saved life.

Case 2A.—A strong healthy man, K., a drayman, aged 30, had acute rheumatism with sciatica and implication of a few joints, chiefly the elbow, on the left side. Ordinary pericarditis then developed, which subsided in a few days; then pneumonia, which involved the greater part of the left lung, and appeared to clear up; then the right lung was similarly attacked. As he had gone so far without any grave symptoms, and as I did not believe that rheumatic pneumonia was as serious as non-rheumatic, I abstained from active interference. I am sorry to say that this final complication carried him off.

Occasionally I have bled a patient once, yet unsuccessfully, as I fancy, because the bleeding was not repeated, as in the following :—

Case 1B.—Mrs. R., aged 42, who suffered from chronic bronchitis and I think from mitral disease also, and who had lately become worn out from sitting up nursing her children with influenza during the epidemic at the beginning of 1890, at last caught the disease herself. At first the attack seemed mild, but pneumonia developed, and I was called up one night and found her with extreme dyspnœa and a very rapid pulse. I bled her to about 16 or 18 ounces with very speedy and effectual relief; so that next morning I found her lying down and comfortable. I was very busy then and unable to visit her again that day. The following night she had another attack of dyspnœa; I was not sent for, and she succumbed. It is quite possible that a repetition of the bleeding might have saved her.

Case 2B.—W. A. M., aged 34, whose occupation was driving a butcher's cart, and who, I believe, drank to excess, had acute pneumonia, which came on with vomiting and diarrhœa, and then a rigor. I saw him on the third day, and found him very ill, with active delirium, hallucinations, &c., which made it difficult to keep him in bed. I bled him to 15 ounces, and he was somewhat better through the night. Next morning he had a violent epileptiform convulsion (I do not know the condition of his kidneys and urine). As his surroundings were very inconvenient, and he could not be properly nursed, he was sent to the hospital, where he died. I think that if he had been bled further he would have had a better chance of recovery.

I might, if I had preserved notes of all cases, mention a few others which died in spite of bleeding; but in which it would, I think, be easy to prove that it was resorted to too late to be of any use.

In some cases of acute pneumonia superadded to advanced chronic Bright's disease, with anasarca and pulmonary œdema, I have been disappointed with the effects of bleeding: possibly it was too late, the lungs being already very œdematous.

I hope the above series of cases will not be deemed too tedious.

I do not put forward all the successful cases as models of how exactly the treatment of such cases should be conducted. In several, I think, the result would have been still more satisfactory if bleeding had been performed more freely, and at an earlier stage. I think, however, that they will furnish some idea of what I conceive to be the best indications for resorting to venesection. Blood-letting has been looked on as the prime means of curing pneumonia from the earliest times. The rule was to bleed at the very onset of the attack, the earlier the better. Still, as Andral says: "It is never too late to bleed in acute pneumonia." Sydenham recommended the abstraction of 10 ounces of blood, repeated once, twice, or three times if necessary. He adds that he has never known the disease to be cured in grown-up persons by removal of less than 40 ounces. On the other hand, he allowed that in certain "epidemic constitutions" very free bleeding was injurious.

The question still remains for solution whether it is best to bleed at the beginning or to wait until symptoms of gravity present themselves. I do not doubt that to bleed at the commencement is perfectly sound treatment. It is recommended on the latest and most excellent authority in Osler's 'Treatise on Medicine.' But if it is practised in all cases at the beginning, many must be bled that do not absolutely require it; not that it is likely to be injurious, but it is not always possible to say at the outset whether any particular case is going to prove a dangerous one or not.

Judging from my own experience, it is generally safe to wait, watching carefully until severe symptoms, or warnings of danger, arrive. The symptoms to be specially watched are sleeplessness, delirium, and undue rapidity of the pulse. In pneumonia the symptoms, instead of being violent first and subsiding into a stage of prostration afterwards, seem to be progressive and cumulative, gradually becoming more intense from the beginning, death or recovery occurring near the climax of the disease. If then, as the disease progresses, the pulse is rising rapidly to a rate which seems inconsistent with safety, or if the nervous system is becoming upset with sleeplessness or wandering, from the violence of the illness, and the patient's condition is becoming oppressed and prostrate, bleeding should be performed without loss of time. It will do no harm, and is almost sure to do good

in the course of a few hours. If the patient complains mainly, as is so often the case, of inability to sleep, it may be promised, almost with certainty, that his desire will be granted in the night following the operation.

As a rule it is not necessary to withdraw much blood at once: 10 or 12 ounces at first, and repeated in a few hours or next day, is usually sufficient.

But it is of great importance not to postpone blood-letting until cyanosis or pulmonary œdema appears. This will almost inevitably be too late.

I should like to make a few criticisms here on some recent writings on the subject. Dr. P. H. Pye-Smith contributed a paper on "The Therapeutical Value of Venesection: its Indications and Limits," to the Royal Medical and Chirurgical Society, vol. 74 (1891), p. 47, in which he summarises 49 cases of all sorts of disease in which he had employed venesection. In some cases the results were satisfactory, temporary relief at any rate being given; in others it failed. I think that several of the cases should hardly be regarded as tests of the efficacy of blood-letting, as the amount drawn was so small as to have inappreciable effects. Venesection is like the bow of Ulysses, and often requires a strong pull to hit the mark. Very few of the cases in Dr. Pye-Smith's list are instances of acute pneumonia, and he draws the following conclusion: "Bleeding is unnecessary and probably injurious in cases of lobar pneumonia." Since then his opinion seems to have undergone some modification, for in Allbutt's 'System of Medicine,' vol. v, p. 129, he says: "It (bleeding) may be used with advantage, 6 or 8 ounces, in the very first stage, if there is much oppression. Later in cyanosis, with a small, feeble pulse, full veins, and distension of the right ventricle, withdrawal of 10 or 12 ounces is a rational procedure, and is often successful in tiding over a dangerous period." I think that to wait for cyanosis is often to wait too long; and if the writer would resort to it earlier, as I suggest, he would speak more favourably of it.

In the discussion which followed the reading of Dr. Pye-Smith's paper, venesection was praised by many, especially those who remembered its use in old times. Dr. S. Mackenzie quoted a recent case in which venesection was performed and repeated successfully in just such a manner as I recommend.

Wherever I have seen reports of venesection having been habitually used in modern times, I find it mentioned in terms of warm commendation.

The cases *par excellence* to which it is adapted, and of which many are unfortunately lost for want of blood-letting, are those of sthenic cases in young, healthy, and robust adults. *The object of bleeding is to change a case from a grave one to a mild one*, and this, if judiciously performed in good time, it is very likely to do, although the pyrexia may not be reduced, or the pulse for a time materially slowed. Respiration is usually soon improved, but it must be borne in mind that the full effects of venesection are not usually visible until a few hours have elapsed.

A good deal of incorrect pathology is often talked, in support of the stimulant, as opposed to the antiphlogistic plan of treating pneumonia, about death being due to weakness and failure of the heart, &c., &c. It is, of course, true that if the heart is already degenerated or overworked, it renders the chance of recovery from all diseases, and particularly pneumonia, so much the less, and a certain number of such cases will probably die in spite of all treatment, but such a condition does not contra-indicate venesection—rather the reverse. Also where pneumonia forms the closing scene to the activities of a broken-down constitution, the chances of recovery, under any circumstances, are not likely to be good.

As I wrote some years ago, I believe that the mode in which death takes place is something as follows :—The blood is inflammatory and increased in viscosity : it passes with greater difficulty through the capillaries, especially those of the inflamed lung. It is less thoroughly aërated, partly for this reason, partly because the inflamed and congested lungs contain less air. The swollen lung offers extra mechanical resistance to the passage of the blood along the pulmonary artery, so that the heart, which itself partakes of the general debility of the fever, becomes unable to empty itself properly, and the right ventricle becomes overloaded and distended. Meanwhile the blood undergoes certain definite changes which we at present imperfectly understand. From the outset, and up to the crisis or nearly so, it is highly inflammatory, and presents the phenomena of slow coagulation and contraction of the clot, which produce the "buffing and cupping" characteristic of inflammatory blood. Towards the time of the

crisis it is disposed to deposit its excess of fibrin, particularly on the walls and valves of the right side of the heart, where it is principally stagnant, causing further embarrassment of the heart, till finally the organ becomes exhausted with the strain, and death occurs from its (usually rather sudden) stoppage, accelerated towards the end by the pulmonary engorgement and œdema consequent on the failure of the heart.

Thus the actual finishing stroke of death is due to intra-cardiac thrombosis: if venesection is delayed till this occurs it must of necessity be too late to be of any use, and when death speedily follows it would be absurdly unjust to put the fatal event down to the credit of bleeding.

Many advances have been made of late in pathology, especially in the field of bacteriology. I look forward with confidence to the time when a deeper knowledge of the causes and processes of disease shall lead to a return to the old traditional methods of treatment, and establish them on a scientific basis, thus harmonising rational and empirical medicine.

Dr. Kingston Fowler (Vice-President, in the chair) commented upon the curious coincidence that a paper strongly advocating the use of bleeding in pneumonia should be read before the Society just when they might be said to be celebrating the jubilee of their liberation from the antiphlogistic method of treating disease. It was round this particular subject of pneumonia that the battle had chiefly raged. It was, no doubt, possible that the pendulum had swung too far in the other direction; but he could not go as far as the author in praise of venesection in pneumonia. Blood-letting had at present a sure place in therapeutics, and it was probably more resorted to now than 15 or 20 years ago. Wherever there was over-distension of the right side of the heart, great relief might be afforded by timely venesection. The antiphlogistic treatment of pneumonia had been carried on for many centuries, and would hardly have been abandoned unless there had been good grounds for so doing. Few of them had had an opportunity of watching it in practice. Dr. Pye-Smith had suggested its use in the very early stages of pneumonia. He himself had never employed it, or seen it employed before there were any physical signs, indeed one did not often have an opportunity of seeing cases in this early stage. In cases of chronic bronchitis and emphysema with dilatation of the right side of the heart, one had the typical condition in which venesection could render great service. He had recently had an opportunity of observing that his own proceedings in a particular case were not thorough-going enough. A man was admitted to the Brompton Hospital with extreme emphysema, dropsy, and interstitial nephritis with signs of pressure on the left bronchus, and bronchiectasis in the left upper lobe. The cyanosis suggested the necessity for venesection, and as its use relieved him very much, it was repeated again and again. Southey's tubes were used for the dropsy, together with incisions, and on one occasion, when incisions

were being made, some large vessel, probably a branch of the dorsalis pedis artery, was wounded, but the fact was not noticed until after the patient's legs had been wrapped in absorbent wool, through which the blood trickled on to the floor. This more copious bleeding afforded him a much greater measure of relief than the smaller venesections, and he recovered and left the hospital. He ultimately died from pulmonary œdema.

Dr. Joll said he had been very familiar with bleeding as a therapeutical measure in his practice in Cornwall 20 years ago, and he had known it do a great deal of good, and certainly it never seemed to do any harm. It was employed *there* and *then* as a routine measure in all cases of acute fevers, and he could remember several cases of acute pneumonia in which he had used venesection to the extent of half a pint or so with great relief, except in one instance. In later years he has substituted free leeching (12 to 20 at a time) over the inflamed area with equally good results, in reducing pain and relieving distress of breathing. This treatment should be restricted to robust patients, and should be applied at the onset of the attack. Generally, he regarded venesection as a valuable remedy which had gone out of fashion.

Dr. Lucas Benham, in reply, agreed that it was valuable in distension of the right heart, but that was not within the scope of his paper. In bronchitis one must not expect too much, because there was often an amount of concomitant capillary bronchitis. He said that if once tried it would thereafter be resorted to by practitioners, and personally he would elect to be treated by that measure. He also said that venesection had been successfully employed in many acute diseases ; for instance, in plague, hydrophobia, and intense forms of malarial fever which were otherwise invincible, and that the method might still be advantageously adopted in such cases.

November 28th, 1898.

A CONSIDERATION OF THE VALUE OF PRESSURE IN THE TREATMENT OF WOUNDS.

By A. Pearce Gould, M.S.

Some apology is needed for my venturing to introduce so trite a subject as the Value of Pressure in the Treatment of Wounds at a meeting of this, or indeed of any, Society of medical men. It may fairly enough be urged that all are convinced on this point, and that pressure is a generally accepted means of securing the better union of wounds. My apology is this, that my own experience has led me to hold that no point of surgical practice is too

trite or familiar not only to justify but even to demand the frequent and careful reconsideration of its value. Are we not all in danger of too readily placing our views on many points among a sacred class of *choses jugées*, and accepting them as articles of faith, when our motto ought to be to "prove all things and hold fast that which is good"? The conditions of our clinical problems are so complex that it is very difficult always to associate correctly cause and effect, and to discriminate justly between the essential and the indifferent. The introduction of the antiseptic method of treating wounds has revolutionised the whole subject, and while it has shown us the supreme importance of the prevention of wound-infection, it has also taught us how small a part our other surgical measures take in securing or in influencing the perfect repair of a wound. By its success in securing asepsis the surgeon's art has been greatly exalted, but in much else one thinks it has simultaneously undergone the reverse process of degradation, and surgical righteousness still consists in no small degree in "walking humbly" with nature. It is easy, like the fly on the wheel, to overrate the part we play in those processes we see wrought out in nature's human laboratory, and sometimes when we hinder rather than help, we may plume ourselves upon our special skill and dexterity! Some years ago I had a very striking illustration of this, which, I hope, taught me a lesson. I happened to see a lady who had been twice subjected to the operation of ovariotomy, and at an interval of 25 years. The earlier operation was performed by the late Dr. Tyler Smith, and his after treatment of the patient was this:—She was lifted out of bed on to a chair every night and morning from the first; her position in bed was changed, I believe, every hour, even at the cost of interfering with sleep; and she was fed every hour from the first, and fed largely. On the second occasion the operator was a distinguished ovariotomist who is still with us. His after treatment consisted in rigid immobility for days, at first absolute starvation and then a very spare diet—the antithesis of the former plan. The lady had strong views on the after treatment in ovariotomy, but the point of chief interest in her case is that the period of convalescence was identical to a day on the two occasions. The restless meddlesomeness of the one surgeon and the masterly inactivity of the other, were alike impotent to affect materially the beneficent healing work of nature! Ambroise

Parè was splendidly right when he insistently urged that it was the good God that healed wounds, but I fear that that doctrine, although two centuries old, hasn't met with the cordial and general acceptance it deserved. May I tell another story? Some years ago a lady took her children to a distinguished physician; he examined them, prescribed for them, and especially directed that they were to have a suet pudding every day, and on no account any milk pudding. Unluckily for her peace of mind this lady sought the counsel of another equally distinguished and experienced physician, who also examined her children and prescribed for them, and specially counselled her to give them milk pudding every day and on no account any suet puddings. In her distress of doubt my friend came to me and asked what she was to do; the children happened to be twins, so I advised her to give milk pudding to one and suet pudding to the other, and watch carefully the effect; the result was that when a system of exchange had been duly established between these twins on a sound economic basis, their nursery became the home of happy, healthy children. But the mother pokes fun at her physicians! We cannot shut our eyes to the great tendency in us all to lay undue stress on minute details which are not of real importance, and to drop unconsciously into a thoughtless routine. Who can doubt that the maintenance of one position for days, so contrary to the wont of nature, is an exaggeration of a wise desire to secure rest, and is greatly overdone, even if it ever need be insisted upon? Can we doubt, again, that in the matter of food we often err in withholding it for too long a time, or in giving it in too small quantity, in accordance with some precept or rule which we have adopted and never reconsidered? The unthinking routine prescription of alcohol for nearly every physiological and pathological state is another very apt illustration of the tendency to which I am referring. I would submit, then, that it is wise to review our positions very often, and be ever ready to question and prove again even our most cherished doctrines. It is in this spirit that I would ask you to consider with me to-night whether the commonly held view of the value of pressure in the treatment of wounds is just and sound. To make my position clear, I will at the outset say that my present view is that pressure is largely ineffectual for the purposes for which it is employed, that it is sometimes mischievous, and can be well and safely dispensed with.

I must say that it is difficult to determine exactly what the general opinion on the use of pressure is. Text-books and manuals of surgery say little about it directly and refer to it only incidentally. For instance, in the very recent surgical text-book of Rose and Carless I find that, writing of the treatment of incised wounds, these authors say :—

"If the wound is dry and aseptic, *and efficient pressure* can be exerted by the dressings it (drainage) can be advantageously dispensed with. An aseptic sponge inserted between the layers of the dressing will form a soft, elastic, and yet firm pad for exerting such pressure."

Speaking for myself, I should say that we use pressure in wound treatment for the following purposes :—

1. To secure the exact apposition of the surfaces of a wound.
2. To arrest oozing of blood.
3. To lessen serous exudation.
4. To prevent the bagging of fluid in recesses of the wound.
5. To restrain muscular action, and in that way promote local rest.
6. To support blood vessels suddenly relieved of pressure.
7. And, later on, to prevent the yielding of cicatrices.

These are the objects for which I have employed pressure, and I have for years tried various methods of carrying it out with greater efficiency. In the large majority of cases the issue has been good, but I have more recently come to the conclusion that this was due not so much to the use of pressure as to those kindly forces of nature to which Parè was never weary of attributing his success. For some time past I have almost entirely abandoned the use of pressure where formerly I should have thought it particularly indicated, and not only have the wounds healed better, but the comfort of my patients has been promoted. I will refer to these cases later on.

It is with pressure as with antiseptics and other methods of treatment—the surgeon's true aim is how not to do harm, rather than how to do good. The less active and positive his interference the better. Time was when the surgeon thought that it depended upon him, and him alone, to protect his patient from the most terrible effects of wound-infection, and that if he failed even to

the extent of admitting one microbe the result must be disastrous, and the patient's tissues fall a prey to the unseen and omnipotent foe. We have learnt better, and have formed a higher and truer estimate of the power of the living body to fight a good fight. Were this not so, the whole human race would long ago have become extinct. In the same way, I believe that nature without our help can accomplish those good ends for which we use pressure in the treatment of wounds, and can do so without those risks and incidental troubles that pressure sometimes entails.

For pressure is not always harmless; used without due care, it may easily lead to sloughing. In such a case as an excision of the breast for cancer, where thin skin flaps are dissected up for some distance, and then sutured with some degree of tension, if pressure is applied with the object of securing exact apposition of surfaces and to prevent the accumulation of fluid beneath the flaps, sloughing is very easily produced. And if the surgeon relying on pressure applies it so gently as to guard against this accident, it is not efficient to prevent entirely the collection of fluid in the depths of the wound. It is easy to see that pressure firm enough to close the open mouths of vessels in a wound must of necessity close all the small vessels in the tissues through which the pressure is exerted, and so it follows that when pressure is used for the arrest of hæmorrhage, to be successful it must be dangerous!

Apart from this danger of pressure, I have been struck with its inutility, as evidenced in certain test cases. For example, take the case of a wound in which, when we dress it for the first time—let us say a week after the operation—we find the edges of the cut skin united, but some fluid blood beneath a flap. I have several times tried in such a case—after letting out the blood, emptying the wound completely, and getting the surfaces in apposition—to secure perfect healing by carefully-adjusted pressure, and time after time I have failed, and found, on removing the dressing, that fluid had again accumulated in the depth of the wound I thought I had so securely closed against such an accident. A drainage tube placed in such a wound removes the difficulty at once, even if only a very light dressing is superposed. I feel sure others must have had the like experience, and I attach importance to these cases for this reason—that we must all have noticed the fact that where this accident in the healing of a

wound occurs at first, renewed oozing of blood is exceedingly prone to occur, and I have known it go on for a week or two. If, therefore, we try to combat this by some therapeutic means and succeed, we have every right to speak of our means as useful; but when we use the means in the first instance and the accident does not occur, we have no right to speak of it as successful, because we have no means of deciding whether there was any tendency to hæmorrhage—whether, therefore, our means played any part at all in the happy issue. That is why I refer to these cases as test cases of the value of pressure. Take another—the bagging of pus. Who of us has not been utterly disappointed at the entire inefficiency of pressure in overcoming this condition! I take it that we never think of relying upon it, but in all such cases resort to counter-opening.

To sum up, my contention is that pressure is apt to be either dangerous or is inefficient, and I will now bring my remarks to a close by attempting to show that when it is used without either of these ill-effects the good result is obtained quite independent of the pressure employed. The evidence on this point is afforded by treating wounds without pressure, and the particular form of dressing to which I am referring is the sealing of the skin edges by two or three layers of antiseptic muslin painted over with collodion, a form of dressing quite devoid of anything worthy of the name of pressure.

There is no need to give details of individual cases, and I will refer to groups of cases only. The first instance is afforded by the wounds left by the removal of tumours, such as lipomata, tubercular glands, and thyroid adenomata. Not only in the simple cases, but when, for instance, one has removed many glands from the neck through one or two limited incisions, the extent of the deep part of the wound being perhaps from skull to clavicle and the total length of the skin incisions under 3 inches, I have found that *if all hæmorrhage is carefully arrested*, a collodion dressing without anything else is a perfectly satisfactory method of wound treatment. After an operation for bronchocele, whether that consist in the enucleation of one or more cystic or solid adenomata, or of the removal of one lobe of the thyroid gland, I find pressure entirely unnecessary—the most perfect results can be uniformly obtained without any resort to it. Now these are cases in which not only are there large potential

cavities in the wound, but in which the effect of muscular action on the wound cannot be wholly excluded.

In many cases of tubercular abscess, where the entire tubercular growth is excised, or after evacuation of the softened parts the active "wall of the abscess" is scraped away, if a dry wound is secured, I find a light collodion dressing over the skin incision is all that is necessary. Excision of the hip or knee can be dealt with in the same way so far as the wound in the soft parts is concerned, and I find that a light dressing on an amputation stump or in a case of excision of the breast gives as good—nay, better—results than a firmly compressing bandage. In abdominal surgery, too, compression can be dispensed with, and with distinct advantage. In cases of mere exploratory incisions, of course we have all recognised that, but in cases of ovariotomy for large cysts, papillomatous cysts of the ovaries with great ascites, and of hysterectomy for large uterine myoma—a simple collodion dressing over the wound is not only as good as, but better than, the mountains of cotton wool and firm bandage that have been and still are largely used. I speak of what I have seen. The fact is that what we have been in the habit of attributing to the influence of our therapeutic measures, our compressing and supporting dressings, has been the result of nature's kindly offices. And when we find nature's powers competent to deal with any pathological condition, we are generally wise to leave the matter in her hands and not rush in like fools "where angels fear to tread." My experience has been that the comfort of my patients has been distinctly promoted by ceasing to apply pressure to healing wounds; nature accomplishes the desired effect so gently and painlessly and so much more dexterously than the surgeon, even with "the elastic pressure of an aseptic sponge."

To put it in one sentence, I would submit to this Society that external pressure is unavailing for many of the purposes for which it is employed, and is not an efficient substitute for the most careful hæmostasis or for drainage of a wound; it is not devoid of danger, does not promote the patient's comfort, and may therefore be discarded in wound treatment.

The PRESIDENT said that the drift of the author's paper had taken him somewhat by surprise, as it was always his custom, after removing the breast, for instance, and clearing out the axilla, to put on a large bundle of wool and a tight binder. Was he in future to leave this off and allow

a potential space to remain between the skin and the side of the chest? Or was he again to put in a drainage tube as he used to do in years gone by? In fact, was he to abandon pressure altogether? He was sure that prospective patients with mammary carcinoma ought to pass a hearty vote of thanks to the author for such a suggestion, for he knew that very firm pressure was not always acceptable. As a case in point, with regard to the value of pressure after operation, he mentioned that a fortnight ago he had occasion to remove an unusually large renal tumour by the loin route, and he gave directions to his house surgeon to apply as much pressure as he conveniently could, and that if after a time the patient complained, he was to pretend to loosen the safety pins. Were they in such cases not to try to obliterate the potential, or, rather, the actual cavity, that was left? If so, what was going to happen? Would the cavity fill with blood or serum? The author had not stated exactly what they were to do, and he would like to know whether in such cases they were to revert to the drainage tube.

Mr. Alban Doran said that four or five years ago he left off strapping after abdominal sections, and relied entirely, as other operators have done, upon the many-tailed bandage; and then for a time he thought he would dispense with the bandage altogether, resorting merely to a simple dressing. However, he found that the change was not advantageous. The more abdominal sections he did the more careful he was not to be too sure about anything. One of the chief aims in after treatment was to ensure the downward passage of flatus. No doubt by over anxiety in overcoming intestinal distension, the surgeon might damage or kill the patient, yet the patient was nearly always better when flatus passed. This of course was well known to Mr. Gould. The flatus passing implied that the functions of the intestinal canal were being restored. Then again, the passage of flatus by relieving distension of the intestines set them moving, and so prevented the formation of adhesions, of which symptoms might only be heard of long after convalescence, or possibly not at all. We knew also that distended intestine with stagnant fæces in it presented a favourable condition for the growth of germs, which, if there were scratches in the peritoneal coat, might escape and produce deleterious effects. He therefore advocated tolerably firm, but not tight, pressure as the best treatment after abdominal section. This was especially the case after the removal of very large tumours, as, for example, papillomatous cysts or free papilloma of the ovary with ascites. He dwelt on a case of a gigantic bilateral growth of the latter kind which looked malignant. After he had removed it, the patient suffered somewhat from flaccid abdomen, but on the second day he put on a bandage and then flatus passed freely. He was of opinion, therefore, that moderate pressure favoured the passage of flatus. It was very important not to apply the pressure only over the lower part of the abdomen, but to make the bandage reach right up to the xiphoid cartilage, otherwise the transverse colon would be pressed upwards and obstructed.

Mr. Stanley Boyd was astonished to hear that the author had used simple collodion dressings with so much success. He had himself long used collodion dressings, together with compressive wool dressing outside the collodion, and, in spite of both, had not infrequently failed to prevent collections of blood in wounds. There were certain places where one soon learned that effective pressure was impossible. Thus after removal of half a thyroid, he had applied such moderate

pressure as could be borne in that situation. In spite of it, oozing took place into the wound, and he was hastily summoned to find the patient all but asphyxiated. He did not think that this accident was due to any carelessness on his part with regard to hæmostasis. The loin was another region where efficient pressure could not be applied. In both regions, unless he felt satisfied that he had checked all bleeding, he should not like to trust to pressure, but would use a drainage tube. With regard to the general cavity of the abdomen and intra-peritoneal operations, he thought that pressure was used for several purposes, some of which had been mentioned by previous speakers. He thought, with Mr. Doran, that the most important was to prevent the intestine taking advantage of the lax abdominal wall to become passively distended. From what he had seen, he thought there was good ground for believing that one could, by the application of a bandage over the whole abdomen, prevent, to a considerable extent, this passive distension of the intestines. He did not suppose that in the great majority of cases pressure was really needed to prevent accumulation of blood in the abdominal veins. He himself had used collodion scabs, if he might judge from what the author had said, even more widely than the author. Thus his practice of late, with regard to amputations of the breast, had been to take a square foot or so of gauze and to collodion that over the whole side of the chest; over this he applied a compressive dressing, and kept the area against the side. Speaking from memory, he did not think he had had a case of oozing under the flaps with that dressing; if any bagging had occurred, it had been at the anterior part of the wound where one could least apply ordinary pressure, *i.e.*, pressure succeeded where it could be well applied: bagging occurred where pressure failed. He had resorted also to another method in order to prevent this accumulation, viz., the buried suture. Many of them would remember how frequently hæmorrhage between the surfaces, and failure to unite used to occur after perineorrhaphy before the introduction of deep stitching. Now, with buried sutures to obliterate the "dead" space, they rarely had failure. In the same way, whenever he got a deep wound, even if that wound only went through the fatty layer of the abdominal wall, or whenever he raised a long flap, he now passed a few catgut stitches so as to obliterate the wound cavity. That method had given very satisfactory results, but, unfortunately, it was not applicable everywhere. If pressure could be abandoned, undoubtedly the patients would gain greatly in comfort; but if its abandonment necessitated the introduction of a drainage tube, he thought that the patients would run dangers which immediate closure of the wound avoided.

Mr. Marmaduke Sheild laid it down as a general safe rule in surgery that there was no one system of treatment applicable to all cases. Although he sympathised with much that the author had said, he thought that there were many cases in which moderately-applied pressure would prove advantageous. In extensive breast wounds it had always been his practice to apply a certain amount of pressure to assist the process of healing. In that way they could coaptate surfaces as one could not in any other way. Then again in the operation for inguinal colotomy, if no pressure whatever were applied and if the patients were to vomit, as they often did, the chance was that some of the small intestine would be found in the bed in the morning. He had seen this happen even in spite of pressure. He referred to the practice of the late Sir George Humphry who used to leave his wounds to nature, and

though not treated aseptically they healed very well. He had long since given up firm bandaging, after amputations, merely applying collodion dressing and leaving in a drainage tube. The crux was to have an open mind in all matters connected with surgery, and not to treat wounds on any fixed principle. The universal discarding of pressure would be a mistake.

Mr. Wallis said he belonged to two bankrupt hospitals, and he thought these were more prolific of dressings than many richer institutions. He thought the safest place in regard to this matter was in the middle. Very likely there were certain cases in which, as suggested by the author, pressure was possibly unnecessary. On the other hand there were cases in which, in spite of pressure, flatus accumulated. That was a common experience. After removal of tuberculous glands in the neck or of the thyroid there was often a certain amount of serous exudation in spite of such pressure as one could apply. He had been much struck by the use of buried sutures. That was carried out by the adjustment of the various tissues when pressure could not be applied on the outside. He thought that if they tried more to approximate the tissues so as to avoid any real or potential cavity the necessity for outward bandaging and heavy compresses would be done away with. With regard to the extraordinary pressure sometimes put upon the abdomen, that was, of course, absurd, because the ordinary condition of the abdomen was constantly changing. He remembered a case of gastro-jejunostomy which he had to leave before the dressing was completed, and his house surgeon put a very strong layer of plaster over the abdomen. The next day the patient complained of pain, so he cut through the bands, but he really thought she would have died, as the abdominal wall advanced fully 6 inches.

Mr. Peyton Beale pointed out that pressure was often employed for keeping dressings in position, and in this manner might be said to help the healing of a wound. It was required when large cyanide dressings were used, especially in the neck, for it was difficult to get such dressings to remain fixed. For this purpose he thought that general elastic pressure, such as could be obtained by the use of a few turns of elastic webbed bandage over a good padding of salicylic wool, was as good as it was easy of application. In favour of the author's view of the uselessness of pressure as a whole, nothing was more striking than the effects of pressure after excision of joints. These did very indifferently if much pressure was applied, whereas they did well if the dressing were applied lightly.

Mr. Pearce Gould, in reply, pointed out that he had purposely limited his paper to one point, the advantage or disadvantage of pressure in the treatment of wounds, and had not entered into the general question of the treatment of wounds. In reply to the President, he should advise him to return to the use of the drainage tube for 24 or 48 hours whenever there was oozing of the blood or the probability of serous exudation. He himself had gone through the same experience of giving up the tube and resorting to pressure, and now he had gone back to the tube. With regard to the case of the removal of a large tumour of the kidney in which very firm pressure was made by the house surgeon, he thought the patient would have been much more comfortable, and the issue of the case quite as satisfactory without the very firm pressure. Mr. Doran had spoken of the value of pressure in abdominal surgery in aiding the patient to pass flatus along the intestine. He himself had had no

difficulty in this matter, without pressure, and he could not at all understand how pressure brought about this happy result. If Mr. Doran had an attack of indigestion with colic, would he button up his trousers tightly? He thought not. Nothing relieved a patient who was suffering from flatus so much as the removal of everything that was tight. As Mr. Wallis had aptly pointed out, the abdomen of all parts of the body was least supported, and was most subject to movements in the natural condition. Every one resented pressure or constriction of that region almost as much as in the neck. With reference to Mr. Doran's case of papillomatous cyst with ascites, he recalled the case of a woman, aged 50, with this condition and extreme distension of the abdomen and papillomatous tumours of both ovaries in which he had operated without applying any pressure. This patient was never distressed with flatus, and left the hospital well three weeks later. He had seen cases in which very large ovarian tumours has been removed and dressed without any external support: the belly wall of its own natural elasticity had fallen back upon the intestines, and the patient had been comfortable from the very first. He agreed as to the value of buried sutures, and he employed them in wounds of the subcutaneous tissues, using fine silk, but for deeper parts he used fewer buried sutures than he used to. With regard to inguinal colotomy, it had never been his practice to apply more pressure than was necessary to keep the dressing in position, and he doubted its value. He pointed out that when a bandage was applied on the operating table with the patient still breathing quietly under an anæsthetic, if that patient vomited the abdominal muscles contracted and left a space into which the intestine could easily pass beneath the bandage if so disposed. In vomiting, the abdominal walls contracted, and the abdomen as a whole became smaller, and any bandage around it must therefore become loose just at the moment when its support was most needed. He thought the escape of small intestine could be prevented, quite easily, by careful suture of the colon to the skin. He was interested in hearing the results of the late Sir George Humphry's treatment of stumps without dressings and, therefore, without pressure. With regard to the effect of pressure in checking serous exudations, he thought it was important to remember that it might be the cause of that condition. The first vessels to be affected by pressure were the veins, not the arteries, and venous compression might cause oozing of venous blood and free serous oozing later on. They must keep an open mind on all these topics, but he was convinced that the purposes for which he had formerly used pressure could be attained just as well without it. If surgeons would only try to do without pressure, they would soon find that they could safely and advantageously dispense with it. He did not wish to say that every wound must be treated with the collodion dressing. Hæmostasis was an exceedingly important point in the treatment of wounds, and drainage was often very valuable; he attached more importance to drainage now than he did five years ago.

December 12*th*, 1898.

TRANSPERITONEAL LIGATURE OF THE ILIAC ARTERIES.

By Bernard Pitts, M.A., M.C. Cantab., F.R.C.S. Eng.

This paper is really an account of two cases of ligature of the iliac vessels—(1) of the internal iliac for pulsating tumour of the buttock; and (2) ligature of the common iliac for hæmorrhage from a pelvic abscess. In both these cases any method of securing the vessels without opening the abdominal cavity would hardly have been adopted by any surgeon of the present time. The advantages of the direct method of ligature seem to me to be so obvious that it is not necessary to discuss them. Connected with these cases are many points of obscurity in diagnosis and of general interest which seem far more worthy of discussion than the particular method of reaching the vessels. I may say in brief that for ligature of the common iliac, internal iliac, and perhaps with rare exceptions of the external iliac, the transperitoneal operation is the one I would choose.

Case 1.—A man, aged 41 years, was sent to me in January, 1894, by Mr. Mennell, of Shepherd's Bush, with a pulsating swelling of the right buttock, which had been first noticed in October, 1893. Five years before the patient had a fall on to his sacrum, and six months later he had sciatica in his right leg, which lasted some weeks. After this he remained well until December, 1892, when he began to have pain in the right leg; and in July, 1893, he complained of numbness as well as pain, and consulted a medical man, who treated him with iodide of potassium. There was a doubtful history of syphilis. On examination, he was found to be an anxious-looking man, complaining of swelling in the right buttock, with pain down the leg and a feeling of numbness. During the last month he had been troubled with irritability of the bladder and pain extending from the anus to the penis. A marked swelling was seen in the right gluteal region, which pulsated visibly. The most prominent portion of the swelling and the greatest pulsation was at a point just external to the right sacro-iliac synchondrosis. The swelling extended from the middle of the sacrum to a point 5 inches external to this, and from the crest of the ilium to a point $5\frac{1}{2}$ inches down; it was firmly fixed to the deep structures. The skin was freely movable over it, and showed no discoloration or glazing. There was no apparent glandular enlargement. Pulsation could be greatly diminished by pressure along the line of the iliac crest. The pulsation was somewhat expansile in character,

and a loud bruit was present. Nothing abnormal was detected in the vascular system beyond a slight aortic systolic murmur. Examination of the chest showed signs of old bronchitis and emphysema of the lungs. My first impression was that the case was one of pulsating sarcoma of the pelvis, and I sent the patient into hospital for observation. During the next fortnight he complained of great pain, and the incontinence of urine and fæces became very marked. There was considerable difference of opinion amongst my colleagues as to the nature of the swelling, and by several it was thought to be aneurysmal in character. Even supposing my view that it was a sarcoma was correct, treatment by ligature of the internal iliac might do good and relieve some of the pain. On February 3rd an incision was made in the middle line of the abdomen, the intestines were held aside with flat sponges and broad retractors, and the posterior peritoneum was divided also in the middle line and reflected to the right, carrying the ureter with it. Search was then made for the internal iliac artery, but without success. To gain more room a transverse incision was made through the greater part of the right rectus muscle. A vessel was exposed below the pelvic brim, which was clearly not the internal iliac, but probably the gluteal. Having failed, as I thought, to find the internal iliac, I exposed and ligatured what I believed to be the common iliac—two goldbeater-skin ligatures were employed—but after drawing these ligatures tight it was found that pulsation still continued in the femoral vessel. Further examination showed that the external iliac artery was lying just outside the vessel ligatured. The common iliac was very short, and the internal was correspondingly long, and lying in almost the whole of its course above the brim of the pelvis, parallel with the external vessel. By ligature of the internal vessel all pulsation in the tumour was arrested. The peritoneum was replaced, and secured in the middle line by a few fine silk sutures. The relief afforded by the operation was at first very marked ; the pain diminished, and the swelling, besides losing its pulsation, seemed to get a little smaller. I was fully convinced that the aneurysmal view was the correct one. In about a week some pulsation was again observed, but it never returned to the same degree as before operation. On the sixtieth day, whilst the patient was turning himself in bed, spontaneous fracture of the right femur occurred just below the great trochanter, and it became obvious that the primary tumour of the buttock must be a sarcoma. The fracture was treated by a long outside splint and extension. He remained in a bed-ridden and distressing condition, but without any evidence of tumour at the seat of fracture, and no increase in the gluteal swelling. The liver showed some increase in size, and just outside the position of the gall-bladder a hard nodule could be felt. On the 158th day after ligature the limb was removed, to give the man a better chance of leaving his bed. The amputation was just outside the capsule of the joint. No evidence of growth was discovered at the seat of the ununited fracture at the time of amputation. Microscopical examination by Mr. Shattock showed a round-celled sarcoma in the medullary canal. Great improvement in the patient's general condition followed, and he was soon able to get about on crutches, and he left for his home in the country 232 days after admission. He came up to see me some six months later in greatly improved condition. The gluteal swelling was larger, and still pulsated slightly ; he had still loss of control over the sphincters. The nodule in the liver was larger and somewhat tender on pressure. He was, however, able to get about and to attend a little to his business, and was much less

troubled with neuralgic pains. He had an attack of congestion of the lungs a year later, from which he died. Unfortunately, a *post-mortem* examination could not be obtained.

When the patient was first under observation the main points in favour of the tumour being a sarcoma were that the pulsation was much more intense in one particular area and that comparatively superficial pressure along the crest of the ileum considerably diminished the pulsation. The swelling was more widely diffused than one would expect with an aneurysm of a vessel like the gluteal at its origin. On the other hand, there was no evidence of bone expansion, as so often occurs in pulsating tumours of bone, and the point of greatest intensity of pulsation corresponded fairly well with the main trunk of the gluteal. The treatment by ligature was followed by decided improvement and I believe delayed the growth considerably in its development and certainly encourages one to adopt this course in cases of tumour unsuitable for radical removal. In ligature of the internal iliac artery it is desirable to make out clearly the common iliac and its bifurcation before hunting for the internal iliac in the pelvis. The abnormality met with in this case is, I believe, not very uncommon, and much time would have been saved if it had been recognised at the first stage of the operation. Certainly the transverse section of the rectus muscle might have been avoided. The behaviour of the spontaneous fracture was remarkable. I have several times seen union occur in spontaneous fracture from carcinoma, but never when it is the result of sarcomatous invasion of the bone. I did not therefore expect to get union when I treated the fracture by splints, but I expected to find evidence of growth around the broken ends and was much surprised when at the amputation no such appearance presented itself. Mr. Shattock, however, on making a microscopical examination of the medullary tissue at the seat of fracture reported that it showed a round- or polyhedral-celled sarcoma with a large number of blood vessels, the latter giving a nævoid condition to the growth. The tumour might therefore be termed a cavenous or nævoid sarcoma. It is interesting to note that although the patient lived for nearly eighteen months after the amputation and that this was done so close to the growth, yet no return took place in the stump.

Case 2. *Hæmorrhage from pelvic abscess treated successfully by ligature of the common iliac artery.*—A man, aged 24 years, was brought to see me in October, 1894, by Mr. Kitching of Cobham, with the following complicated history. He was perfectly well till the age of 14 years; he had then whilst at school in Scotland in February, 1886, a boil on his neck attended with fever and some pleurisy of the right side. He was two months in bed, and towards the end of this time he had an abscess in the right lumbar region which was incised and which discharged for a few days. Some contraction of the left leg was then found and extension was ordered. He returned home to Liverpool and was seen by Mr. Mitchell Banks, who treated him with continued rest for an inflamed psoas. Mr. Banks informs me that he could find no evidence at that time of any mischief in the vertebræ or pelvis. After some months' continued rest in a leather splint he was taken for a sea trip, and he got apparently quite well. He returned to school after an absence of eight months, and in 10 days the leg was again giving trouble—viz., soreness and pain—and he had to return home and remained away from October, 1886, to May, 1887. In August, 1887, his right shoulder became painful and an abscess discharged spontaneously in October at a point corresponding to the insertion of the deltoid. The arm was kept to the side for six weeks and the sinus healed, and he remained perfectly well till October, 1888, when pain and swelling recurred in the shoulder and an incision had to be made, and the arm had to be kept quiet for eight weeks. In the following June an incision was again necessary. He left school in July, 1889, and, except for most obstinate constipation, he remained well till October, 1893. He was at Oxford from 1890 to 1893. In 1893 he went to America, and on the voyage he had sciatica of the right leg, and during his stay of two months the condition of constipation greatly increased. In June, 1894, he was again troubled with pain down the right leg and in the loin and along the iliac crest, with increasing difficulty with the bowels and occasional feverish attacks with a rise of temperature. He was an only son; his parents were alive and very healthy. The patient was a very tall, strong-looking man, and beyond a scar in the right loin and one in the right shoulder nothing surgical could be detected. I referred him to my colleague, Dr. H. P. Hawkins, and under treatment he made some improvement, and was able to return to business in May, 1895. In July he suddenly developed another attack of pain in the legs and tenderness in the groin, and a little thickening and resistance were to be felt at the outer part of the iliac crest. He afterwards went to Ireland and was very well for a month there. In October he again had pain down the leg with fever. In January, 1896, he went to India, returning home in April. He was in considerable discomfort during the whole trip, with great irregularity of the bowels and feverish attacks accompanied by pain about the right hip. At times he noticed some blood and mucus with the motions and thought that an abscess was discharging per rectum. He had much pain when straining at stool, and was unable to walk straight on account of discomfort about the groin. In July, 1896, he went into a nursing home in London, and the symptoms seemed to point to the appendix as the seat of trouble. He had a tender, deep swelling which felt very hard in the iliac region, below and outside the usual portion of the cæcum, with a temperature varying between 99° and 101° F. The evacuations were carefully watched and with the decline of the temperature, the relief of the symptoms, and the diminution of the swelling, there was a definite

discharge of pus in the stools. It was now clear that he had a deep-seated abscess which periodically discharged into the bowel, and both Dr. Hawkins and I were convinced that the appendix was the cause of the trouble. So definite a discharge of pus had taken place and with such a marked improvement that operation was decided against. He returned home and was carefully watched by Mr. Kitching, who sent him again to the home in London on August 24th, as he then had some hardness and redness of the skin over Poupart's ligament. An incision was made and a small quantity of thick pus was evacuated (about 2 or 3 drachms). A long sinus was traced from the incision just outside the femoral vessels under Poupart's ligament into the pelvis, but no bone could be felt by a bullet probe. It was now clear that the abscess was independent of the appendix and most probably was due to some obscure bone trouble in the lumbo-sacral region. A drainage tube was inserted. The after progress was satisfactory ; there was very little discharge in the dressings and the temperature became normal. He left for his home in three weeks' time. On October 23rd I saw him again with Mr. Kitching as he had had a temperature for several days ranging from 101° to 103°, and again a good deal of tenderness about the sinus and in the right iliac region. I dilated the sinus and introduced a larger tube ; the discharge was of a thin unhealthy character. Warm boric fomentations were ordered. The temperature fell to normal, but on October 27th —*i.e.*, two months after the operation—he suddenly had severe hæmorrhage from the sinus, which was stopped by pressure and a tight compress. On arrival I found him blanched and with a rapid pulse and constitutional signs of loss of blood. The mattress was soaked with blood. The compress was removed and blood began to come freely from the sinus, and it evidently came from the deeper parts of the abscess cavity ; compression of the common femoral or the epigrastic or circumflex iliac with the finger in the wound did not arrest it. A careful plugging of the cavity with iodoform gauze was done under chloroform. The following day the patient's temperature was 104·4°, but it gradually came down and all went well for a week, the gauze plug being changed on the fourth day. On November 5th I was again sent for as the hæmorrhage had recurred and the patient was in a very exhausted and critical condition. Mr. Stabb saw the patient with me and I was prepared to ligature the internal iliac artery. The position of affairs was difficult, for the patient's father, who was just returning from America, was expected in a few days' time. The wound was carefully replugged and it was decided to remove him in an ambulance by road (a distance of 20 miles) to the St. Thomas's Home for Paying Patients. Mr. Stabb and Mr. Kitching kindly travelled with him. During the next few days he improved in condition. On the morning of November 15th severe hæmorrhage again took place, which was stopped by the resident medical officer of the home with firm pressure over the gauze plug. On arriving I repacked rapidly and determined to operate without further delay. The father had in the meantime returned from America and both he and the patient were wishful for radical measures to be adopted.

The operation took place on November 15th at 2 p.m. The anæsthetic was given by Mr. Tyrrell and I had the advantage of the able assistance of Mr. Stabb, Mr. Abbott, Mr. Edmunds, and Mr. Kitching. The condition of collapse was such that we expected to have to infuse during the operation. A free central incision was made from just below the umbilicus to the pubes. The colon was examined, and it was found that

the appendix was normal, but that the large bowel was very elongated and the sigmoid flexure had a very large omega-shaped curve, thus accounting for the great trouble for so long a time with the evacuation of the bowels. An incision was made in the peritoneum over the medial line of the sacrum, and the internal iliac artery was exposed without difficulty. The gauze plug could be felt extending behind the peritoneum to within an inch of the main trunk. The vessel was found to be very fixed by chronic inflammatory products, and all attempts to pass an aneurysm needle failed. I was particularly anxious to ligature the internal vessel, since owing to his anæmic condition I was fearful of the circulation in the limb if the common iliac were ligatured. An attempt was therefore made to pass the aneurysm needle behind the artery and vein and so to ligature *en masse*. A considerable gush of blood took place from a wound of the vein, which was arrested by pressure on the common iliac. The attempt was therefore abandoned, and the common iliac was exposed and a stout floss silk ligature passed and the vessel tied by a surgeon's knot. We found that the circulation was not controlled, the ligature being too thick for the hitches to be efficiently tightened. The vessel was then tied in two places with a thinner floss silk ligature with an interval of $\frac{1}{2}$ inch between them. The original thick ligature was then removed. The circulation was now effectively controlled. The peritoneum was adjusted but not sutured, and the operation was finished as quickly as possible. The patient took the anæsthetic remarkably well, and it was not found necessary to infuse. The abdominal wound healed without trouble and the warmth of the limb was well maintained, though pulsation in the tibials was not re-established for some weeks. A remarkable change took place in the chronic abscess lining—it necrosed and a very offensive discharge of a free character continued for several weeks. Rapid repair then set in and in six weeks the sinus was soundly healed, and has never given any trouble since. During many of the short febrile attacks which the patient had had between 1894 and 1896 he often had pain and considerable tenderness in the right shoulder and several times I expected suppuration to occur there, but these symptoms always passed off with the improvement in other conditions. He remained in St. Thomas's Home for seven or eight weeks and left for his own home greatly improved in condition. The right leg was diminished in size, but the collateral circulation was well established. He shortly afterwards changed his residence for a more bracing atmosphere, and has since been under the care of Dr. A. Lyndon. The patient wrote to me on November 15th, 1897, being the anniversary of the last operation, and expressed himself as feeling extremely well and putting on flesh at such a rate that all his old clothes had to be let out; he was moving about in the house freely without crutches, but using them as advised when out of doors. The bowels were still a difficulty and the use of enemas was frequently necessary. Occasionally he had spells of soreness above the hip, but much fewer and milder than formerly. His general health was perfect. In December, 1897, I was asked by Dr. Lyndon to see him for a swelling at the lowest part of the abdominal cicatrix. The swelling was reducible and had some impulse, and it was difficult to say whether it was a hernia or an abscess presenting through the cicatrix. The swelling was a little tender, and there was a sense of fluctuation. It was covered with gauze and wool and a few days later burst and a large quantity of laudable pus escaped into the dressings. Careful examination of the sinus showed that it led deeply into the pelvis but without

apparent connection with the former abscess cavity, the cicatrix of the old sinus remaining perfectly sound and free from tenderness. He was kept at absolute rest by Dr. Lyndon, and in March, 1898, the floss silk ligature presented itself in the discharges. After this the sinus slowly closed and has been for some months soundly healed. I last saw the patient in London and ordered him to wear a truss for a weakness of the lower part of the cicatrix. He was in very good health; he weighed 14 stone and was apparently at last free from any inflammatory condition.

The points for consideration in this case seem to be as follows: 1. The original infection. I believe it to have been tuberculous. We have pleurisy as an early symptom, and this was followed by several suppurative troubles, most probably connected with superficial caries of the humerus just outside the joint. The affection of the left psoas which was so obstinate can be best explained by similar bone trouble low down in the vertebra. We have then a long period of intermission and again chronic inflammatory trouble in the pelvis affecting the right side. It is not likely that any inflammatory affection other than a tuberculous one would have remained latent so long. 2. The diagnosis between inflammatory trouble due to chronic appendicitis and that due to bone disease is sometimes very difficult. Looking back on the case I think that more attention ought to have been paid to the pains down the leg which appear so often in the history but were never at all prominent whilst the patient was under observation. One prominent feature in the case was the constant association of discomfort and slight rise of temperature with constipation and the relief afforded by laxatives and enemas. 3. Hæmorrhage from chronic abscess ulcerating into vessels is usually significant of bone trouble, and occurs perhaps most frequently in popliteal abscess due to caries of the back portion of the femur. The recurrent and sudden outbursts of bleeding in this case pointed to a communication with a large vessel, and was of quite a different character from the bleeding sometimes met with from unusual vascularity of the granulations. I do not know of any case where ligature of the internal iliac or the common iliac has been necessary, but am confident that this patient's life could have been saved in no other way. 4. The behaviour of the ligature in this case was unusual. After lying apparently quiescent for a year it set up suppuration and was eliminated. For a large artery I believe the stay knot suggested by Mr. Ballance and Mr.

Edmunds in their work on the 'Ligature of Arteries' is the best. I have used it several times and cannot now explain why I did not employ it in this instance. My attention was so occupied with the other circumstances of the case that I probably did not give this point full consideration at the time. In tying the artery, however, I endeavoured to stop the circulation with the least amount of constriction and I believe that I did not rupture the coats.

The PRESIDENT asked whether the second case might not have been one of those rare, latent forms of septic disease. He also asked why the author had employed a goldbeaters'-skin ligature in the first and not in the second case. He questioned whether that material had any real advantage over the old-fashioned silk ligature. Personally he preferred a substance for ligatures which was always at hand, and he knew of no material better than silk. He admitted that in some cases the silk would find its way out, but was not that also the case with the other? He asked the author what he thought was the connection between the large pulsating tumour on the right buttock and the sarcoma of the shaft of the femur. Was the one secondary to the other, or were they parts of a continuous growth? He thought that one result of the paper would be that when a surgeon found a pulsating tumour in the buttock, the exact nature of which he could not actually determine, he would not stand by waiting and consulting, but would proceed to tie the artery at once, in the hope that the tumour was, after all, non-malignant. And even if it did prove malignant no harm would have been done. He sincerely congratulated Mr. Pitts upon his successful treatment of what was evidently an extremely anxious and difficult case.

Mr. HARRISON CRIPPS observed that anything like a transverse incision in the abdomen was a very unsatisfactory thing, because it was very difficult to get accurate junction and proper union. In the author's case, however, it certainly appeared to have been necessary. It would have surprised him very much if the ligature which had been handed round had not made its appearance in the sinus. He was quite clear that the absorption or otherwise of a ligature depended much upon its size. This particular ligature was so thick that it was impossible to conceive of its being absorbed. He asked why the author had used such strong silk, adding that if he had used number 1 or 2 it would probably have been absorbed. He knew from experience that in ovariotomy if one used a thick strong ligature it would often turn up again, whereas with a fine well boiled ligature nothing more might be seen of it. The President had suggested that it was an operation which should be undertaken in any case of pulsating tumour of the buttock whether sarcoma or aneurysm. He himself, however, would require some strong evidence to induce him to perform the operation. As to the details of the operation he recalled a case of his own of aneurysm of the external iliac artery in a man of 60. The patient was a big corpulent man weighing 16 stone. He presented a pulsating swelling in the groin, which, two months previously, was the size of a walnut, but had since attained the size of a cocoanut. He was a sporting man, and had tried to "ride it off." Of course, it was usually easy enough to feel the common iliac arteries,

so he had set to work thinking he would have no difficulty in securing this vessel. He made his incision on the left side of the rectus muscle. He could not take it as low down as he wished because of the swelling. On passing in his hand he could feel the pulsation of the vessel, but was surprised at the extreme depth at which it was situated. He enlarged the incision and endeavoured to secure the vessel by getting at it from the inner side, but, after trying for a quarter of an hour, it was evidently quite impracticable to get at it in that way. The intestines crowded over it and occluded the field of vision. He then turned the sigmoid flexure to the right, and got at the vessel from the outer side. The operation then became a comparatively easy one. He wished to emphasise the point, because if he had made his incision in the middle line the change of tactics would not have been possible. He regretted to say his case did not turn out as satisfactorily as the author's, possibly because the intestine had been very much mauled during the operation. In any future case he would be inclined to make the incision as in this case, and attack the vessel from the outer side.

The PRESIDENT remarked that he had not suggested that the surgeon should ligate the iliac artery in a case of sarcoma, but that instead of standing by the bedside unable to make up his mind, it might be the right thing promptly to apply a ligature—if only as a means of diagnosis.

Mr. MARMADUKE SHEILD congratulated the author especially upon his second case, which he said had only been brought to a satisfactory result by an exhibition of the greatest surgical skill. With regard to the nature of the case, he would be inclined to class it as one of what Sir James Paget used to call "chronic pyæmia." He referred to a case of a man who, after an attack of supposed influenza, got suppuration within the antrum; in 18 months an abscess opened in the axilla and neck, and the pus, in that instance, contained staphylococcus aureus. Presumably, the organ had been lying all that time in the glands. In chronic abscess of the breast, the indurated parts often broke down into abscess years afterwards. Passing on to consider the method of ligature employed by the author, he recalled that in a letter to the 'British Medical Journal' in 1892, he had advocated direct ligature of the iliac vessels through the peritoneum. Soon after the publication of that letter, he received a communication from a well-known Army surgeon who said he had tried it and had found it very easy to perform. He himself was a great believer in the silk ligature, soft, not corded. That held in a way that no animal ligature would. He thought that if the vessel were healthy the best plan was to draw the ligature tightly enough to injure the inner coat, as securing the best result, sealing by plastic inflammation.

Mr. BALLANCE, referring to Mr. Sheild's advocacy of the transperitoneal operation in 1892, mentioned, without wishing to put forward any claim to priority, that in 1890 Mr. Edmunds and he had described and illustrated the transperitoneal ligation of the great arteries of the abdomen. The operation in a very fat person was no doubt a difficult one, but they had explained the best method of retracting the intestines in their paper. The incision they recommended was directly over the course of the artery. He did not quite understand why the author had made the incision through the posterior peritoneum in the middle line. He thought that the parietal peritoneum having been fully exposed, the most direct method of getting at the artery would be the best. The nature of the ligature used was a question which particularly interested

him, as he had made numerous experiments in this connection. He agreed that floss silk was one of the best ligatures; the silk, in fact, which Mr. Pitts had used for his operation. Floss silk fulfilled every indication required of a ligature. He did not agree with the strictures that had been passed on the size of the ligature, for No. 1 silk might cut through the outer coat of a great artery, and even in the hands of skilful surgeons bleeding had occurred from that cause at the time of the operation, or soon after. With regard to the goldbeaters'-skin ligature which was introduced by Mr. Edmunds and himself, he still thought that it was a very good one. It was of course in principle the same as Lord Lister's peritoneal ligature, his idea being that the best ligature would approximate in structure to the tissue of which the outer wall of the artery was composed, and peritoneum was almost entirely composed of white fibrous tissue. Goldbeaters' skin was only peritoneum which had been beaten out to an extreme thinness. He had found it easier to deal with the goldbeaters' skin than with the crude peritoneum. Goldbeaters'-skin ligatures could now be obtained of any size; they were very strong, round, and inelastic. A No. 5 or 6 would last in the body 150 to 200 days, which was long enough for any purpose. It was true that silk could be made aseptic by boiling, but there was no harm in having a choice, and it was possible to have a better ligature than silk. They had at St. Thomas's two specimens of silk ligature of an interesting character. One was an ovarian pedicle which was ligatured 18 months before death, and nothing but the silk knot was left, the loop having been absorbed. There was another ovarian ligature in the museum applied three years before death, and the most careful dissection of the pedicle showed that there was no ligature left at all. One might say, therefore, that a loop of silk would disappear in 18 months, and that in three years even the knot would disappear. Encapsulation occurred, but this was only part of the process of the absorption. It was not extraordinary that the ligature had come away in Mr. Pitts's case, he had been obliged to apply it in the neighbourhood of the abscess cavity, and under these circumstances there was a great opportunity of its becoming infected. With regard to the real nature of his last case, whether chronic pyæmia or tuberculosis, it hardly appeared possible to come to a conclusion. He did not think that, as a rule, chronic pyæmia could extend over such a long period with such slight manifestations. Cases had been reported before that Society in which tuberculosis of glands had involved the great arteries of the lower limb. He suggested that the original condition of the groin was tuberculosis of the glands remaining latent for a long time and then breaking down and involving the vessels.

Dr. John Brunton pointed out that much of the silk sold as pure was really a mixture of silk and jute. This was easily detected on burning the sample. He did not believe that the ligature which had been passed round was of pure silk. The fibres of jute under the microscope were very like those of silk, except that the latter were separate while those of jute were in bundles.

Dr. Lyndon, in reference to the nature of the case, said that in the history the patient had given him the abscess of the shoulder which he had at school was diagnosed as pyæmic, and when the abscess burst through the abdominal wall the pus was examined for micro-organisms especially tuberculous, but he had only found pyogenic organisms. There was no doubt that the abscess was caused by the ligature, because

at the time the abscess burst the sound went down to bare bone on the inner surface of the horizontal remus of the pubes. After that the sinus changed its direction and went back towards the iliac synchondrosis where the ligature had been applied. On removing the tube one day the ligature was found in the mouth of the tube. It was undoubtedly a large ligature, but it had undergone a very great change in appearance in the bottle it was preserved in. Since it came away the sinus had slowly but steadily healed, whereas previously there had been no attempt at repair. Personally he regarded the case as one of tuberculosis.

Mr. Bernard Pitts, in reply, said he had no very positive opinion as to the cause of the pelvic abscess, but his own feelings were strongly in favour of a tuberculous origin, from the fact that he did not know of any septic condition which could remain dormant for so long a period as was covered by these successive small explosions in other parts. He pointed out that the size and weight of the patient were not incompatible with this conclusion, and tuberculous affection of glands might occur in persons of excellent physique. Some of them doubtless fell victims to acute tuberculosis, but many went on with nothing but the scars to show what had happened. He had a still more open mind with regard to the ligature. He was prepared to use any ligature which could be warranted free from germs, and he would prefer one which would last for a long period. He admitted the criticism with regard to the apparent size of the ligature, which, however, had changed considerably in size and appearance since it was used. He thought a large soft ligature was best for a large artery, and less likely unnecessarily to damage the coats. He was quite convinced that traumatism of the coats was not necessary, and that repair could be successfully carried out without it. With regard to the case of sarcoma, he thought the tumour of the femur was secondary to the growth in the buttock; at the same time one must remember that pulsating sarcoma was apt to occur in a number of bones almost at the same time. He recalled a case in which there was a loud bruit and dulness in the chest, and it was thought there was an aneurism. That opinion was held for some time, until a pulsating swelling formed low down in the lumbar region which established the diagnosis. He agreed that it was desirable to avoid a transverse incision of the muscular wall of the abdomen if possible, but if the surgeon wanted room he must have it. That was the case in the present instance. He carefully sutured layer by layer, and no hernia had followed the operation. In respect of ligature of arteries, there was no difficulty as regards the internal iliac with a central incision. To ligature the common iliac it might be better to make the incision over the outer border of the rectus. The common iliac on the left side was a difficult artery to expose, especially in a stout patient, but he would be inclined to go straight down through the mesentery until he reached the vessel, rather than run the risk of too much handling of the intestines.

January 9th, 1899.

THE CONDUCT OF THE HEART IN THE FACE OF DIFFICULTIES.

By Sir Wm. Broadbent, M.D., F.R.C.P.Lond., F.R.S.

Mr. President, I should like the Society to understand that I am in no way responsible for the title of this communication, but that I accepted it at your dictation. There are appeals more imperative than commands. Such was yours when, as an old pupil become president of this Society, you requested me to read a paper on "The Conduct of the Heart in the Face of Difficulties." I was bound to obey, but I ventured to point out that the word "conduct" in this phrase might have two distinct meanings—the behaviour of the heart itself under difficulties, or the way in which it might be helped and guided through them by the physician, and to ask which of the two questions was to be discussed. You promptly answered "both." It is by your command, therefore, that a *double entendre* is inflicted upon the Society. In dictating the title you practically also decided the way in which the question was to be treated.

The difficulties which the heart has to face are sometimes of its own making, sometimes imposed upon it from outside. An over-distended stomach, for example, pushes up the diaphragm and presses upon the right ventricle; effusion into the pleural cavity, or more rarely a mediastinal tumour or aneurism, carries the heart to one side or other of the chest; or the flow of blood towards the right auricle may be impeded by pressure on the superior vena cava. Difficulties of another kind may arise from obstruction in the arterioles and capillaries—in the pulmonic circulation by emphysema or by acute affections of the lungs; in the systemic circulation by the numerous conditions which give rise to high arterial tension.

The heart adjusts itself to altered conditions in a wonderful fashion. It may be so much displaced by effusion into the left pleural cavity or by traction from a shrunken right lung that its beat is felt as far out as the right nipple and yet we can trace very little inefficiency in the functional action. Time, however, is

an element in this accommodation. The same degree of displacement occurring suddenly, as in pneumothorax, will be attended with severe dyspnœa, and great disturbance of the action of the heart will be occasioned by sudden and capricious distension of the stomach.

The difficulties arising out of flatulent distension of the stomach or colon or intestinal canal generally, will require some attention, since they are the cause of most of the functional derangements to which the heart is subject, and give rise to the heart complaints which occasion in the aggregate perhaps more suffering than does actual heart disease. The heart often tolerates a considerable degree of upward pressure of the diaphragm, and it is not uncommon to meet with stomach resonance as high as the fifth space, and to find the apex beat displaced upwards and outwards to the fourth space and outside the nipple line, without conspicuous symptoms. But the heart behaves very differently in different subjects in the presence of flatulent distension of the stomach. It partakes of the general constitutional condition of the individual; in the strong, therefore, it is vigorous, in the weak it cannot be anything but weak. Moreover, the heart has very special relations with the nervous system; it reflects every emotion, beats high with courage, is palsied by fear, throbs rapidly and violently with excitement, and acts feebly under nervous depression; but it is not only through the cerebro-spinal system that the heart is influenced, it is in immediate relation with the vasomotor nervous apparatus, and in a scarcely less degree with the sympathetic system generally. Normally, afferent impulses are constantly flowing from the viscera to the central nervous system, and by this reflex process their blood supply is regulated, and their functional activity is governed. These afferent impulses when perverted by functional derangement or disease may become serious disturbing influences. The nervous system in a large and increasing proportion of people is unduly sensitive and excessively mobile, and the reactions to influences of every kind are exaggerated. In some a little emotional excitement gives rise to palpitation, and a piece of bad news or the bang of a door seems to stop the heart altogether. There is in such subjects no form or degree of cardiac disturbance which may not be caused by indigestion, scarcely any symptom of cardiac disease which may not be simulated. Add a touch of hysteria on the lookout for symptoms

and for someone to give ear to the narration of the unparalleled agonies of the sufferer, and the difficulties of the heart, and it may be added of dealing with them, are complete.

It is of course of the greatest importance that we should be able to distinguish these functional affections of the heart from troubles due to organic disease, and this is especially the case where there is severe pain in the cardiac region. The absence of physical signs of valvular or structural change will be a help, but murmurs may be present at one or more of the orifices during palpitation when there is no valvular affection, and there may be actual mitral or tricuspid incompetence when all the symptoms are really of neurotic or dyspeptic origin.

Angina pectoris is one of the cardiac affections which may be closely simulated by the effects of dilatation or functional derangement of the stomach. The first question to be put in a case of cardiac pain of anginoid character is "As to the circumstances under which it comes on?" Whether as an effect of exertion or during repose? The earlier attacks of true angina are practically always provoked by exertion, while spurious angina is specially liable to come on during repose. It is true that angina when established may come on in the night, or may be induced by the act of undressing and the contact of cold sheets, but there will be a history of attacks during exertion. Pain and a sense of suffocation may also be brought on by the pressure of the abdominal viscera reinforcing that of a distended stomach on lying down, whether the heart is diseased or sound, and a weak heart may actually be brought to a standstill in this way. Speaking generally, angina pectoris in a woman is always spurious, and the more minute and protracted and eloquent the description of the pain the more certain may one be of the conclusion. Again, when palpitation or irregular action of the heart, or intermission of the pulse, or pain in the cardiac region, or a sense of oppression follows certain meals at a given interval, or comes on at a certain hour during the night, there need be little hesitation in attributing the disturbance, whatever it may be, to indigestion in one or other of its forms. Nightmare from indigestion is not a bad imitation of true angina. So also if any cardiac symptom or pain can be walked off, it may usually be set down as functional, and due to some outside disturbing influence or to nervous irritability. The same may generally be said of intermission of the pulse, of which

the patient is conscious, and, though with less confidence, of irregularity of the heart's action—if the patient feels it the irregularity is usually temporary, and not the effect of organic disease.

In these functional affections, it is not the heart which is to blame; it is more sinned against than sinning, and if its difficulties are removed there will be nothing to find fault with in its conduct. The difficulties are, as has been said, the state of the nervous system, on the one hand, and of the digestion on the other, and according as the neurotic or the dyspeptic element predominates will be the treatment required. No details need be entered into, but one observation may be made. Patients suffering from these functional derangements of the heart usually make them a pretext for avoiding exercise, and often for taking stimulants or drugs, whereas exercise and fresh air are what he or she most needs. The best way to prevent the expenditure of superfluous energy on the part of the heart in the form of palpitation is to give it a fair amount of legitimate physiological work to do; and to relieve one attack of palpitation or faintness by alcohol is to invite another, while the terrible danger of drifting into alcoholism is incurred.

One of the most common difficulties with which the heart has to contend is high arterial tension, or rather the obstruction to the onward movement of the blood in the capillaries and arterioles which is the cause of the high pressure in the arteries. While dyspeptic troubles and other reflex sources of irritation give rise merely to functional affections of the heart, high arterial tension when persistent is a frequent cause of actual disease. The resistance in the peripheral circulation has to be overcome and the heart rises to the occasion. It puts forth the increased energy required and in doing so becomes hypertrophied. Hypertrophy is not disease, though sometimes the heaving impulse and powerful throb of the apex are complained of by the patient and looked upon with suspicion by the medical man, but the development of additional muscular fibre is accompanied by the development of increased connective tissue, and when in the decline of life the nutrition of the more highly organised structures is no longer vigorous, the fibroid element may gradually predominate over the muscular, or fatty degeneration may take place.

But the valves may suffer before the muscular walls. Where greater force is required to propel the blood into the aorta there

is greater strain upon the mitral valves during systole, and a more violent recoil upon the semilunar aortic valves during diastole. This gives rise to chronic inflammation of the valves, with thickening and contraction, and, in the long run, insufficiency.

It was necessary to mention high arterial tension on account of its frequency and importance as a source of cardiac difficulty, but I have dealt with it so often and so recently that I will forbear from further dwelling upon it on the present occasion, only remarking that the recognition of unduly high pressure in the arteries affords one of the most valuable indications for treatment in a great variety of conditions.

I am afraid it often escapes recognition, and sometimes digitalis is given for the relief of the cardiac discomfort which may attend it. This is like knocking the head against a stone wall, for digitalis not only acts on the heart, but tightens up the vessels, and so increases the obstruction, already too great.

Coming now to the serious difficulties to which the heart is exposed by reason of damage to one or other of its valves, we discover, say, a systolic murmur at the apex or at the right second intercostal space, indicative of leakage of the mitral valve or of interference with the blood current at the aortic orifice. What are we to do? Frighten the patient out of his life or out of his peace of mind? Condemn him at once to live on one floor, and forbid him exercise and excitement and all that makes life tolerable, and give digitalis? Certainly not. Or, shall we ignore the murmur on the chance that it may not be serious, which is a not uncommon proceeding when a medical man has predicted sudden death once or twice, and found the patient to go on living for 10 or 20 years? This would be equally unreasonable.

The first thing to be done is to ascertain what the murmur really means; whether, when it is mitral, there is much or little regurgitation, or, if aortic, whether it signifies mere roughness or actual construction. Numerous considerations enter into the determination of these questions, of which we need specify only those arising out of the conduct of the heart. If, in the case of mitral incompetence, there is any considerable reflux into the left auricle, the first effect will be damming back of the blood entering it by the pulmonary veins, and the obstruction thus created will make itself felt in the pulmonary artery, raising the blood pressure

within it. There is no branch of the pulmonary artery on which we can place our finger or a sphygmograph, but the high pressure is at once accused by accentuation of the pulmonic second sound. If the circulation is to be maintained under these circumstances, something must be done to overcome the obstruction in the pulmonary circulation and neutralise the mitral reflux. This can only be by increase in the capacity and strength of the right ventricle. The right ventricle accordingly becomes dilated and hypertrophied, and the dilatation and hypertrophy which we call compensatory become for us the measure of the regurgitation. This is the conduct of the heart in the face of this particular difficulty, and we learn from the amount of compensatory change required to neutralise the effects of the valvular lesion whether the lesion is severe or slight; our conduct, then, will be guided by the degree of efficiency of the compensation. When there is no appreciable hypertrophy of the right ventricle or marked accentuation of the pulmonic second sound, and the patient has no heart-symptoms, the murmur means nothing, and there is no need to interfere in any way with the patient's mode of life even if this include hunting or climbing, or swimming, or cricket. I should draw the line at football or training for races of any kind.

If with marked hypertrophy and dilatation there is still no breathlessness on ordinary exertion, or other symptom of disturbed circulation, the regurgitation is considerable, but it is neutralised by the compensatory changes. We are not called upon to do anything, but the patient must be warned that the compensation may easily be broken down, and that a single imprudent act of violent or sustained exertion may do irreparable injury.

Cardiac symptoms, such as breathlessness on slight provocation, show that the compensation is inadequate, and it is only by great carefulness that the serious effects of the valvular lesion can be put off. Let us suppose that we have the heart landed in extreme difficulties from incompetence of the mitral valves, the liver enlarged till its lower border crosses the abdomen at the level of the umbilicus, the veins of the neck distended and pulsating, the face and lips livid, the lungs congested, the legs dropsical, the urine scanty, turbid, and albuminous, the patient gasping for breath and unable to lie down. It is in such a case that digitalis and the like remedies find their opportunity. But first

the right side of the heart must be relieved from the over-distension which is paralysing its efforts. Unless this is done, the digitalis may simply help the straining ventricle in the work of self-destruction. The nearest approach to a modern therapeutic miracle is seen on bleeding in a good case of this kind. When I say a good case I mean one in which the onset of the severe symptoms has been sudden, under the influence of some adequate exciting cause, such as over-exertion or chill, in a fairly robust subject with a powerful right ventricle. The venesection must be followed up by a good calomel purge—2 or 3 grains of calomel, with, say, 5 grains of colocynth and hyoscyamus—and perhaps a dose of white mixture. Bleeding, however, is too heroic a method for these degenerate days, and it is not always easy to say whether it is really demanded. A good alternative is six, eight, or a dozen leeches over the enlarged liver, followed up, of course, by the calomel purge. In less severe cases we may content ourselves with the mercurial aperient alone.

The right heart having been relieved, digitalis may be given with excellent effect in different combinations, according to the condition—with nux vomica and ammonia, and perhaps ether, or with acetate of iron and potash. If the œdema is considerable, the fluid should be drained off by Southey's tubes, and any pleural effusion should be withdrawn by aspiration at an early stage.

Mitral incompetence will serve as an illustration of the difficulties imposed upon the heart by disease of the valves. They differ in the different valvular affections, and the heart responds in a special way for each one. It is unnecessary to go into details with regard to all of them, and the time at my disposal would be quite insufficient. The principle which I wish to emphasise is, that when the heart is in difficulties we can generally give more relief, indirectly, by removing these difficulties, than directly, by aiding it to overcome them. This is the case whether the disturbing influence is external to the heart—as, for example, a dilated stomach, or distended colon, or resistance in the peripheral circulation—or is a secondary effect of disease of the heart itself, as illustrated by over-distension of the right ventricle. To take another instance, if the heart is in a state of fatty degeneration, it is useless to give cardiac tonics, its work must rather be diminished by keeping down the arterial tension, and a fatal issue may be for a time averted by preventing

distension or dilatation of the stomach. Such illustrations might be multiplied indefinitely.

When, therefore, we are considering the treatment of cardiac disease, the first question to engage the attention is how we can relieve the labouring or harassed heart by the removal of some condition which is causing or aggravating the difficulties with which it is already contending. In doing this we often put an end to the symptoms which have given rise to suffering and anxiety, and in all cases we make the action of digitalis or other cardiac tonics more efficacious.

The PRESIDENT remarked that he felt sure that if their late friend Dr. Hare could have been present, he would have entered a gentle protest against the expression made use of by the author that "bleeding was too heroic a measure for these degenerate days." He asked whether the days had actually degenerated or even the practitioners. He confessed that he would feel somewhat depressed at hearing Sir William suggest that practitioners were at fault in this respect; personally he had a high opinion of the value of venesection in the case of the overloaded right heart.

Dr. SANSOM, alluding to the tolerance of the heart in respect of wounds, mentioned that only the other day he heard of a case in which the right ventricle had been opened by a spear wound, was stitched up, and the patient recovered without a bad symptom. He had also read the account of the *post mortem* of a person who died at the age of 70, in whom a bullet was found embedded in the apex of the heart; certainly there was nothing to complain of in the heart's action in that case. They knew very well that wounds of the heart, moreover, were not usually productive of much suffering. At all events, the heart could stand interference very well, whether by the knife of the surgeon or a body embedded in its tissue. He pointed out that stitching up a wound in the ventricle entailed certain changes of an inflammatory nature (partial myocarditis), yet recovery might take place. Did not that show that the musculature of the heart *quâ* muscle was not of the supreme importance that had been thought? Yet the heart *quâ* heart seemed occasionally to behave very badly under comparatively trivial circumstances. He suggested that they ought to think more about the nervous system of the heart and less, though sufficiently, of its muscular apparatus. As a muscle it was not more important than any other muscle provided that the mechanical difficulties were overcome. One met with one form of inflammation of the heart of grave importance and with others that did not mean much. As to fatty degeneration of the heart muscle, he recalled that in replying to a question on that subject by the late Dr. Fothergill he had confessed to him that the state of his mind "was one of hopeless imbecility." If he wanted a good specimen of fatty degeneration of the heart he sent to the *post-mortem* room for one from a case of anæmia, pernicious for choice, yet if that patient had recovered from his complaint that heart would have got quite well again. In the anæmic the heart was sometimes dilated, sometimes shrunken, sometimes large, sometimes small, but the patients generally got well. Then, again, inflammations of the walls of the heart were very irregular in their ways and habits;

some got perfectly well, some made the heart hopelessly irregular and it stopped to beat no more. He mentioned these things because he thought the muscle of the heart had been unduly blamed for the physical conditions associated with it as in dilatation of the heart or other misbehaviour. It was not a question merely of muscle but of nerve and muscle. As a matter of fact they knew nothing of the heart muscle apart from the nerve elements. When they talked of restoring compensation, or of helping the heart over certain difficulties, it was not a mechanical question of stimulating the muscle, but of turning the scale to get over certain difficulties. It was too often the case when the heart was irregular that the practitioner said : "Let us give digitalis." It did good and it was persevered with ; but it was possible for such a patient to take too much digitalis, or for too long a time, with, he feared, in some cases most unsatisfactory results. If one could only tide over the primary difficulty and follow that up by good nutrition of the heart and, by healthful measures, restore physiological activity, disaster might be averted. He thought digitalis and similar drugs were sometimes to be credited with sudden death from heart disease. In respect of venesection when practised *ad deliquium*, that was a thing which the present age did not brook very well, but when practised in slight degree in heart disease, sufficient to relieve the tension in the right ventricle, it was in a large number of cases productive of extreme benefit. That had been his experience in both hospital and private practice. Without this method of treatment their control over the heart and its conduct would be small indeed.

Dr. Theodore Williams referred to two very severe cases in illustration of heart difficulties due to a distended stomach. One of them was very striking, the stomach became exceedingly distended, and it was suggested to wash it out. The patient became absolutely livid, and apparently in great danger of her life. The author had seen the case with him and had suggested that it might be due to distended stomach, and that if this condition were relieved the symptoms would probably pass off. This view proved to be right, for the patient soon recovered and lived for some time after. The cases of heart neurosis belong to a very different category. He had seen cases which alternated with asthma in which he had been unable to make out whether there was organic disease or not. The rate of beat and the symptoms generally were so striking that one could not help thinking that there was something more than was apparent. One patient, a medical man, had this pseudo-angina and suffered severely, but he lived for 15 years after the attack, though he died suddenly at last. Both these groups were perplexing. He was glad to hear the author say that he was prepared to bleed in some cases of heart disease. He himself had recommended it several times, but the medical men had told him that it was impossible, and he had to fall back on leeching or cupping. He could not recall more than five or six cases in which venesection was actually done. As to the tolerance of the walls of the heart he had seen some instances of needles in the heart, and curiously enough when the needle was removed no symptom followed. He recalled the case of a little boy in whom in the left second interspace he found what proved to be a needle and this was removed by a surgeon, no symptom following. He had brought a case before the Clinical Society in which an assegai had gone right into the heart. The weapon was eventually removed and the man recovered.

Dr. Alexander Morison asked the author whether he had absolutely satisfied himself that non-synchronism of the ventricles was an established clinical fact. He ventured to think it was at least doubtful. The treatment of heart disease, as managed by physicians, removed from medicine to some extent the opprobrium of ineffectiveness which surgeons at times playfully sought to cast upon it. There at any rate physicians could honestly say that they could materially alter the situation for the better in many cases. He recalled the case of an old friend of his with ascites and a dilated heart, a feeble pulse, and the kidneys blocked by albuminuria and sugar. Surgeons were in attendance with instruments for tapping the abdomen, and the patient implored him to "save him from those Philistines!" By giving large doses of strophanthus, within a week all his serous cavities were emptied, and he lived two years longer. He agreed that too much was done in the separation of the factors in the action of the heart. The functional unit was triune: nerve, muscle, and blood, which worked together to produce a common result. They must therefore, for therapeutic purposes, distinguish which was the predominant influence in a given case. Alluding to venesection, he remarked that the smallest quantity of blood that relieved the heart was about 4 ounces, or about a heartful, and he recalled a case in which that quantity was removed periodically for a length of time, less than that not producing the same benefit.

Dr. Symes Thompson observed that when considering "the conduct of the heart under difficulties" the first thing we notice is that functional disorders are associated with great perturbation, whilst organic defects may cause but little apparent discomfort. Palpitation, fluttering, faintness, &c., are marked in the first case, but are often absent in the second. In the one case the patient is sure that his heart is diseased, in the other he will scarcely admit that anything is wrong. When the derangement is in the nerve supply, the agitation is more than commensurate with the importance of the lesion, but where mechanical defect exists, the heart adjusts itself to its extra labour and the discrimination and caution of the physician are needed to prevent undue strain. In treatment it is of more importance to relieve the circulation than to stimulate the heart, just as in kidney disease we must relieve the overtaxed organ, not by stimulating it to greater exertion, but by inducing other organs, *e.g.*, the skin and bowels, to do its work. If we stimulate the heart or the kidney we may increase the dangers, but by removing the strain we save our patient.

Dr. Maguire spoke of a cause of difficulty which the heart had to encounter which had not received adequate attention, viz., the negative pressure in the thorax, which was increased during inspiration and diminished during expiration. The negative pressure was at once an assistance and at times a hindrance to the circulation, an assistance always, a hindrance only in disease. In health, of course the increase of negative pressure during inspiration helped to draw blood from the large veins, and might perhaps assist in bringing about diastole of the heart. The increase of negative pressure during inspiration might be diminished in disease, as for example in emphysema. A very small amount of emphysema made breathing very short. He himself attributed this to a deficiency of the movements of the chest walls which lessened the suction action of blood to the thorax. The pulsus paradoxus was only mentioned once in the discussion on adherent pericardium last year, and then only to be thrown aside. It had been found that the symptom was not peculiar to adherent pericardium. It must, however, be a symptom of something,

and it was, he thought, due to the hindrance which the increase of negative tension in the thorax during inspiration offered to a weak left ventricle. If one watched persons dying, with gasping respiration, and a heart with absolutely no reserve power, the pulsus paradoxus would be found, *i.e.*, absence of pulse during inspiration. They could produce it in themselves by throwing the shoulders back and the arms behind, and stretching so as to increase the diameter of the chest. One might sometimes see it with a patient sitting quietly in the chair without any exertion at all. He thought it would be found that many cases of what was called irregularity of pulse were instances of this, each beat corresponding to inspiration being wanting.

Dr. Calvert referred to senega, which was described as a stimulating expectorant, *i.e.*, that it stimulated the heart and circulation. It was a fact well known to physiologists and pharmacologists that senega had a very decided depressing action upon the heart. Yet it was used in chronic bronchitis, and the later stages of acute bronchitis—that is in conditions where the right ventricle was failing or was likely to fail—and of pneumonia as a stimulating expectorant under conditions where a depressant of the heart was above all things contra-indicated. There was about half a grain of the active principle in an ounce of the infusion, and if this were prescribed every three hours—as it often is for days together—in 24 hours the patient would have taken 4 grains of the active principle. If 4 grains of the active principle were injected into the circulation of a fairly strong cat the animal would be killed at once by arrest of the heart; 15 grains of chloral hydrate similarly injected into the circulation of a cat would not necessarily kill it, so that 4 grains of the active principle of senega were much more depressing than 15 grains of chloral hydrate. Moreover, the drug was an irritant to the stomach, and as it was prescribed for the most part in conditions where the stomach was passively congested—on this account also the routine giving of the drug was to be deprecated.

Dr. Robert Jones referred to the relation between the heart (in the absence of organic disease) and certain toxins, notably those of fatigue products caused by severe epileptic convulsions. He instanced the case of a man subject to general convulsions, who on one occasion was seized with Jacksonian epilepsy to the number of 40 a day. He was unconscious and bedridden, but his temperature remained normal and the pulse 50 until the epilepsy became general, and with the advent of the status epilepticus his temperature rose to 103° and his pulse to 140. During the Jacksonian fits the accumulation of fatigue products was insufficient to raise the temperature or disturb the heart, but the moment the convulsions became general the heart became affected. He would be very glad of any light upon these cases from Sir William Broadbent, as he had lately several fatal cases of the kind, and in one bleeding and digitalis had been tried with disappointing results.

Dr. H. A. Caley observed that failure of compensation in some cases was difficult to account for by any increasing or protracted mechanical strain, and must be attributed to failure on the part of the myocardium, in the causation whereof toxins might play a part. He suggested that the element of interference with the coronary circulation was concerned in the difficulty which some chronic heart cases had to face, and was operative before any effects directly attributable to degeneration of the coronary arteries were manifest. Myocardial weakness, whether of toxic origin or consequent on defective nutrition, might play as important

a part in producing heart failure as mechanical embarrassment of the circulation, even in cases in which the latter factor was the primary one.

Sir WILLIAM BROADBENT, in reply, said that it was a fact that venesection was not employed to anything like the degree it ought to be. There was nothing in one's experience to compare with relief to an over-distended right heart afforded by bleeding. He could not agree, however, that 4 ounces would be sufficient. This quantity of blood removed from a vein when there was a ligature between it and the heart could hardly afford much relief to that organ. He himself took 14, 16, or even 20 ounces. As to frequency, he had not had occasion lately to practise it much, but very few of his house physicians had gone through their six months without having to bleed on more than one occasion. The only surgical instrument he carried about with him was a lancet, because he found that the general practitioner often had not seen it done, and had not with him the means of doing it. As to the number of times it could be employed with safety, he recalled the case of an old lady between 80 and 90 who had been bled 52 times, and she lived to over 90. She was a typical high-tension old lady who, not in spite of her bleeding, but by reason of it, had lasted so long. Dr. Sansom's remarks about injury to the heart reminded him of some he had seen. He had once seen a long needle transfixing the septum, probably done during infancy, and there it had remained for many years, the patient dying of erysipelas. In a case which was related before the Clinical Society, the right ventricle had been punctured under the impression that there was an effusion into the pericardium, and the patient survived. He had ventured to suggest, under the circumstances, that possibly the patient had survived in consequence, but he was sharply reprimanded by the then President for being frivolous. He did not think that fatty degeneration from degenerated or blocked coronary arteries was the same one got in anæmia; indeed, there was very marked difference in the consistency of the heart-muscle in the two conditions. He concurred in the view that interference with the coronary circulation was an element in production of cardiac trouble long before the stage of degeneration. When one was dealing with aortic valvular disease which comes on late in life, his apprehension with regard to prognosis was not based upon the changes in the valve, but upon the apprehension that these changes might extend to the commencement of the coronary artery, and thus determine functional inefficiency, and then degeneration of the myocardium. He agreed that a weak heart might be brought to a standstill by dilation of the stomach. He recalled a case in which the cause of death was clearly of this kind. The patient was an old gentleman leading a very active life, who one day experienced a feeling of great discomfort. This sensation had recurred a few days later. When he saw him the stomach was extremely distended, much displacing of the heart giving rise to weakness and irregularity of the pulse. His condition was decidedly alarming, and he had advised the patient to go home at once, instead of which he went to the City, ate a hearty lunch, and then went a considerable distance into the country. He dined late and heartily, and after dinner he went upstairs, but he had no sooner laid down than he died. As to non-synchronous action of the ventricles, this might apply either to duplication of the first sound or where there were two beats of the heart to one of the pulse, both conditions being fairly common. He still believed that in both instances there was a want of accord between the two ventricles. When one had an opportunity of listening to these cases,

without saying that there was an absolutely alternating action, it was quite certain that in the beat which did not reach the pulse the left ventricle must, if acting at all, be acting very feebly. That was especially evident when there was a systolic mitral murmur that was only audible in connection with the beat which reached the pulse. He referred to the interesting case of a man who had fallen down in the street in a fit. When he saw the patient he was breathing very rapidly, with a very irregular pulse, full bounding and large, 20 to 30 in the minute. On listening to the heart, he found that to every pulse there were five or six beats which were clearly of the right ventricle only, and when the left ventricle beat came it did not coincide with any of the right ventricle beats, so that evidently the two were acting independently. His explanation of the pulsus paradoxus was not that of Dr. Maguire. He did not think that it was the action of negative pressure upon the left ventricle, but on the left auricle. He could not imagine that any degree of negative pressure would stop the ventricle contracting, but by acting on the auricle it might prevent the ventricle filling.

January 23rd, 1899.

THREE CASES OF APPENDICITIS AND DIFFUSE PERITONITIS.

By Cuthbert S. Wallace, F.R.C.S.

The subject of diffuse peritonitis is of great interest, both on account of the diversity of opinion that prevails about its treatment and the extent and nature of infection from which recovery is to be looked for.

A few years ago an English writer said that he doubted if a single case of general peritonitis was saved by surgical treatment.

This gloomy view is refuted by many published cases, as well as the three following, which I think are of sufficient interest to bring before the Society:—

Case 1.—E. T., aged 17, labourer. The patient, who gave no history of any previous illness, was attacked with sudden pain in the right side of the abdomen on July 21st. The pain was at first only slight, but on July 23rd so increased in intensity that the patient applied to a hospital for advice, and was sent away with some medicine. In the early morning of July 24th the pain still further increased in severity, so that the patient again sought medical advice, and was admitted at 2 a.m. by Dr. Sykes. The abdomen was rigid all over, but not distended at all. Tenderness was general, but most marked in the lower right lateral portion of the abdomen. The lower part of the abdomen was motionless on respiration.

The pulse was 100, the tongue dry and slightly brown, and the temperature 103°. The bowels were open the day before admission; there had been no vomiting. The abdomen was resonant, except over an area immediately internal to the right ant. sup. spine extending upwards, downwards, and inwards for the space of 2 inches. The right loin was dull nearly up to the costal margin. The abdomen was opened through the right rectus below the umbilicus. The intestines were injected, and between the coils there was much sero pus. The peritonitis was general. The appendix, perforated at its base and containing a concretion, was found lying to the inner side of the cæcum, and adherent at its apex to the omentum. Around the appendix was a foul abscess whose walls were formed by the omentum and cæcum. The appendix with the adherent omentum was removed. The intestines were then turned out on to the surface of the abdomen, flushed with water, and the lymph, as far as possible, removed. The abdominal cavity was cleaned with irrigation and sponging. A tube was inserted into the cæcal region, and a gauze drain into the pelvis, and the wound sutured in layers. The recovery was good except for a fæcal abscess that discharged on the third day, but quickly healed up.

Case 2.—F. S., aged 25, parlourmaid. On two occasions, three and six months before admission, the patient had suffered from mild attacks of appendicitis. During the night of July 23rd she was seized with violent pain in the chest, which became rapidly diffused over the abdomen. Vomiting occurred several times. On July 24th she got up and waited at breakfast, but was soon obliged to give in and go to bed. When first seen at 4.30 p.m. on the same day the abdomen was rigid and tender, the breathing almost entirely costal. The eyes were sunken, the pulse 130, and the temperature 103°. As there was some suspicion of a perforated gastric ulcer, the abdomen was first opened in the mid line immediately below the ziphisternum, but the stomach and anterior aspect of the transverse colon and great omentum were healthy. A second incision was then made through the centre of the right rectus, below the umbilicus, and evacuated thin sero pus. Here the evidence of peritonitis was unmistakable and involved all the viscera behind the great omentum. The appendix was found lying on the pelvic brim surrounded by foul pus, which also extended into the pelvis. The intestines in the neighbourhood of the appendix and in the pelvis were covered with thick lymph. The appendix, inflamed and presenting a sloughing point at its apex, was excised. The intestines were turned out on to the abdomen and washed over to remove the lymph, and then returned to the abdomen after this had been cleaned by douching and sponging. Gauze drains were then inserted into the pelvis, and the cæcal region and the wound closed in layers. The temperature quickly fell to normal, and the general condition rapidly improved. The patient was discharged on August 20th with all the wounds sound.

Case 3.—J. W., male, aged 40, was admitted on July 23rd with the following history:—Five years ago he suffered with a severe attack of abdominal pain, which was mostly localised on the right side. Since that time he had remained well until the present illness. This commenced on July 20th with sudden pain and tenderness in the abdomen. He did not seek advice for this until the day of his admission, when he was unable to empty his bladder. The abdomen was then found to be

distended, rigid and tender, with little or no movement on respiration. The right iliac fossa and loin were dull. The liver dulness was raised to the fourth rib in the nipple line. The patient's expression was anxious, and he complained of violent pain in the abdomen. The pulse was 120, and the temperature 102°. McBurney's incision was adopted, and the appendix sloughing at its base found in a foul abscess that lay below and to the outer side of the cæcum. On gentle traction the appendix came away, but no proximal end could be found to which a suture could be applied. Towards the mid line no limitation of the abscess could be found, the intestines being congested and contained blood-stained sero pus between their coils. The abdomen was therefore opened through the right rectus sheath, when it was found that the whole of the intestines were injected, and had lost their polish. These were accordingly turned out on to the surface of the abdomen. Great difficulty was experienced in dealing with the coils in the pelvis, as they were adherent, covered with lymph, and intensely congested. It was now found that the abscess extended up along the ascending colon, and had gained the upper surface of the right lobe of the liver. The intestines were washed over with hot sterile water, and the lymph removed. The abdominal cavity was flushed with jugs of water, sponged dry, and as much lymph as possible removed from the flank and upper surface of the liver. Drains were placed in the pelvis, and the cæcal region and the wound closed. The temperature fell to normal the next day, and the remainder of the recovery was uneventful.

Putting the cases together, we find that the duration of the symptoms before admission were two days, 18 hours, and three days. These times represent the duration of the local attack, as the histories gave no idea of the date of the general infection. In two instances there had been previous attacks.

In all three cases the features were pinched, the pulse rapid, the temperature raised, and the abdomen rigid, practically motionless and tender.

In two cases the abdomen was distended. The clinical signs of peritonitis were therefore well marked.

Local abscess was present in every instance, and invaded the pelvis in one, the loin in the second, and the pelvis, loin, and subdiaphragmatic space in the third. In no instance was there any definite barrier between the abscess and the rest of the peritoneum.

In the case of the woman the peritonitis had not reached the anterior surface of the great omentum, and was limited in an upward direction by the root of the transverse meso-colon.

In the other cases the peritonitis was general, as evidenced by the loss of polish, injection of the gut, and presence of sero pus.

It is remarkable that the intestinal wall was still thick and that

the bowels in every case responded easily to an enema given on the third day.

The appendix was situated to the inner side of the cæcum, on the pelvic brim and to the outer side of the cæcum in the respective cases.

The organ was perforated at its base twice and at its apex once. In all cases the treatment was practically the same.

The appendix was removed, the intestines turned out on to the surface of the abdomen and kept warm with a stream of hot sterile water while the lymph was removed.

The abdomen was meanwhile flushed out and then gently sponged, especial attention being paid to the pelvis and loins.

The incision through the right rectus below the umbilicus and the turning out of the intestines is, I believe, the best method of dealing with these cases of local abscess with diffuse infection of the peritoneum.

Firstly, the abdomen can be quickly opened; secondly, the appendix can be rapidly found and removed; thirdly, the same opening can be used to cleanse the abdomen; and, fourthly, the wall can be rapidly and efficiently sutured in layers.

Careful attention to the loins I think very important.

When the patient is lying on the back, fluid in the abdomen tends to collect not only in the pelvis, but on either side of the spine about the kidney, right lobe of the liver and hepatic flexure of the colon on the right side, and about the spleen and splenic flexure of the colon on the left.

If great attention is not paid to these parts and especially to the right loin, which from its situation is more often infected than the left, there is grave danger of the formation of a subdiaphragmatic abscess with all its attendant evils.

Of the two rival methods of treatment, namely sponging and irrigation, I think that irrigation is to be preferred, but the irrigation, to be effective, should be combined with exventration of the intestines. While the intestines are in the abdomen it is impossible to direct the water to the exact part required, and the stream out of the abdomen is not sufficiently regular to ensure the infective material being carried away; it is, indeed, in great measure simply washed from one part of the abdomen to another. The complex coils of the intestine render it impossible to be sure that the water has reached every part of the gut.

The method of sponging each coil as it is drawn from the belly means that much time is taken and that a coil that is clean is put back into an abdomen that still contains pus.

Sponging without irrigation must produce more damage to the serous lining if so employed as to in any degree effectively clean the cavity, even if it can be effectively done while the intestines are in the abdomen. In none of these cases was there any difficulty in reducing the bowels, but if the distension was great it could be reduced by multiple puncture.

A drain into the cæcal region is indicated if the abscess formation is well marked, but I doubt if either a tube or gauze drain into other parts of the abdomen is really of much service, as the matting about either soon prevents any save a very local drainage.

Rapidity of operation is an essential and is best obtained by exventration combined with irrigation.

The wound can be drawn together with a few sutures and the permanent closure of the wound left for another day if thought desirable, or the wound can be simply plugged with gauze and left open if time presses.

The prognosis in cases of peritonitis after operation is, I think, very difficult. The local condition is no sure guide as far as the amount of fluid or lymph is concerned, nor does the injection nor loss of polish of the serous membrane materially help. It is believed that the fluids pass out of the peritoneal sac by the lymphatic stream, through the diaphragm, and therefore an infection of the upper part of the abdominal cavity is more likely to be quickly followed by sign of general toxæmia than is an infection lower down in the abdomen. This being so, a peritonitis that has reached the upper surface of the liver, and perhaps formed a subdiaphragmatic abscess, is to be more feared than one that still remains localised to the lower segment of the belly.

This accounts for the extreme gravity of the pus under the diaphragm, though even this is sometimes recovered from without any special drainage, as in the case of the man above related.

The distension of the intestines is unfavourable, as this means that the septic products have produced a profound effect on the general system, for it is probable that the poison acts through the general system and not immediately on the wall of the gut, as the same symptom is seen in septic conditions in which the peritoneum is not infected. In a paper on "Acute Dilation of the Stomach,"

read before the Clinical Society by Dr. Box and Mr. C. S. Wallace, it was pointed out that there was ground to believe that this disease was also produced by some poison. In reference to this point it is to be noted that in the three cases described in this paper the intestines were still thick and opaque. When this is found the prognosis is much more favourable than when the intestines are thin, bloodless, and translucent.

Taking everything into consideration, however, there is no doubt, I think, that by far the most important signs are the pulse and facial expression. Not because they in any way denote the amount of peritonitis present, but because they show the amount of septic absorption that is taking place from the peritoneum.

My best thanks are due to Dr. Turney and Mr. Pitts, under whose charge the patients were.

Mr. Clutton congratulated the author on his success in these three cases, probably a larger proportion than any surgeon in London could boast of at such an early period of his professional career. He could remember a time when exventration was done and not a single case succeeded. Then the dry method was tried without much success, and he was inclined to think, on the whole, that they ought to give another trial to exventration, a point which the paper forced on their notice. They used to turn out the whole of the intestines into a warm wet towel, then flush with fluid, then turn the patient over and allow the fluid to run out. There was always much shock, and that was probably why it did not succeed. The author appeared to have made a small incision, whereas his own incision for exventration was a large one. He asked how the author managed. Possibly also in former days part of the damage was done by the use of antiseptics instead of normal saline. He asked whether the patient was turned over. The same remark applied to operations for injury where peritonitis was not present, in respect of the damage done by antiseptics. He noticed that in every case the author was successful in getting the bowels to act, this often being a point of great difficulty, though most of them had lost patients long before that stage had been reached. Sulphate of magnesia was generally rejected, and it was a real consideration how to get the bowels to act in cases of peritonitis. He remarked that an American surgeon had suggested that in every case in which the abdomen was opened for peritonitis they should inject with a hypodermic syringe a dose of salts at the uppermost point, and this would act in about 12 hours. There was no doubt that toxin poisoning went on from the mucous membrane of the intestinal tract, as well as from the serous surface. With exventration there ought to be no difficulty in injecting a purgative at the point where the small intestine came off from the spinal column. He remarked that the author seemed to have operated early, but that was a point which should be encouraged.

Mr. Stanley Boyd said the subject was one in which he had long taken a special interest, and until lately he used to report annually upon published cases of diffuse suppurative peritonitis. He, however, could never satisfy himself that any of the cases reported as diffuse peritonitis

were actually so. In the cases just read the evidence yielded by ventration was as precise as could well be obtained. He presumed that in the case where the inflammation was found to extend to the upper surface of the liver the author must have enlarged his opening. He asked for further information concerning the author's procedure. He himself had never turned the intestines out for diffuse peritonitis, having been deterred partly by the bad records of the past and by having more than once seen operations of this kind done and witnessed the enormous difficulty of getting the distended intestines back again. Often in order to effect reduction small holes were made in the intestine with a trocar or hypodermic syringe, which not unfrequently leaked on withdrawal of the instrument and had to be repaired, lengthening the operation considerably. Nowadays, one would make a longitudinal incision and thoroughly empty the intestines at once, letting them drain themselves not only of gas, but also of their toxic contents. He had asked himself in the past whether something could not be injected hypodermically to cause the bowels to act in cases of distension with vomiting; he had asked his colleague, Dr. Mitchell Bruce, for a purgative drug which could be used hypodermically; but there did not seem to be such a one. He suggested that it might be worth while to study this question. Further, he had been deterred from turning out the intestines by the practical impossibility of thoroughly cleaning the cavity of the abdomen. Even on the *post-mortem* table it would evidently take an enormously long time to do anything of the kind. It seemed to him that when recovery took place in cases of this kind very much must depend upon the powers of resistance of the patient whatever the method of treatment; that, in short, more credit was to be attributed to the patient than to the surgeon.

Mr. Bruce Clarke said that in order to overcome the difficulty of evacuating the intestinal contents he had on several occasions made an opening into the intestine, sometimes stitching the edges to the abdominal wall. If one were successful in emptying the intestine the patient's recovery was usually very rapid. He had sometimes turned out the whole of the intestines, though by no means in all cases, and he was inclined to think that it was a good thing. He pointed out that it was impossible in such a case to make an abscess cavity absolutely aseptic. What one did was to get away a considerable amount of the materies morbi and so relieve the patient, and the vitality of the patient gradually destroyed the remainder of the poison. He thought the incision through the rectus was a good one, as it left a sounder scar and less tendency to hernia. He asked the exact length of the incision that was necessary to get at the under surface of the diaphragm. He had seen and read of a good many of these cases, and was doubtful as to how far they could truthfully be described as diffuse peritonitis. Most of them were what one would call localised abscess with a certain amount of general peritonitis spreading from it in various directions.

Mr. Battle said he had recently been discussing the question of operation in these cases with a physician who seemed to think that it was useless when there was general peritonitis. He was enabled to tell him of a certain number of cases of his own and had since told him of the author's cases, and he trusted that physician was a convert. He had suggested to the physician that perhaps he had been unfortunate in his cases, which possibly had not come before him early enough for operation to be of use. One hesitated to approach this subject as it

was so very large, and, moreover, it was so much a matter of individual judgment in each case. He referred to the case of a patient admitted on the previous day who had been under observation by a medical man for two days suffering from supposed intestinal obstruction. This case had not been considered urgent, and he did not really look bad, had no mental confusion, and answered questions clearly. The pulse was 100, the abdomen a little distended and hard, but not particularly tender. Closer examination showed, however, that there was a little dulness in the right flank, respiration was entirely thoracic, the pulse flagging, the nostrils working, and there was a bluish tint about the face ; moreover, he had recently vomited. The temperature evening of admission was 102°. He had made an incision in the right side which gave exit to a lot of stinking pus, not entirely localised. The patient's condition even then was such that he did not feel justified in adopting measures that would have meant a larger opening and cleansing of bowel and peritoneum. It was very difficult to treat these cases on any one plan. He added in conclusion that, as far as one could judge, not one of the author's cases would have got well had surgical procedure not been adopted.

Mr. John H. Morgan (the Vice-President in the chair) said the discussion had taught him the value of the incision along the outer border of the rectus. It enabled one to view the appendix and it had the advantage that by extending it one could easily reach other parts should this be required. Turning out the intestines was a somewhat startling novelty, but he had learned his surgery in pre-antiseptic days. Do what they would by sponging or flushing, a certain amount of deleterious matter would certainly be left behind for the patient to dispose of. Much the same conditions of things obtained in perforating gastric ulcer. Yet if the contents of the stomach were not very septic early closure of the opening warranted a very fair hope of a satisfactory result. The author laid great stress upon the pulse and the expression of the patient, but personally he attached more importance to the actual condition of the abdomen, and if thoracic respiration was exaggerated with rigidity of one or both sides of the abdomen, he took the prognosis to be grave.

Mr. Wallace, in reply, said that in the first case he had used MacBurney's method in order to get the appendix away. Although it could be easily removed in this way it was necessary to make a second incision to the outer side of the rectus, the bottom being on a level with the anterior superior iliac spine, going up for six or eight inches. With that incision, by pulling the abdominal wall away, one could see the upper surface of the liver which, in one case, he found covered with thick lymph. He admitted that his were favourable cases because in none were the bowels distended. In neither case, except that of the man, was there much trouble with the intestines. In a subsequent case, that of a young child, he had had considerable trouble in getting them back, and they had to be incised and became rather congested. The two adult cases he certainly did not expect to recover, the facies being very bad and the pulse barely perceptible. As to exventration the incision was made and held widely open and then the small intestine and transverse colon were drawn out while the opposite loin was flushed and wiped dry with sponges, the process being repeated on the opposite side to cleanse the opposite loin. The right flank was much easier to deal with than the left. The

intestines were kept warm by keeping hot sterilised water running over them. If after this procedure one was going to get recovery the improvement usually set in very rapidly, and within 12 hours the abdomen was moving, which he took to be a sign that the operation had done some good. He adhered to the idea that pulse and facial expression were, as far as prognosis was concerned, the most important things to go by, more important in fact than distension or non-movement of the abdomen. The abdomen might be moving perfectly well and yet be full of pus. He had sewn up a duodenal ulcer the other day and the patient went on well for four days, the abdomen moving, and food being taken. The patient then suddenly sank, and his abdomen was found full of pus.

TWO RECENT CASES OF SUCCESSFUL OPERATION FOR IMPACTED STONE IN THE URETER.

By P. J. Freyer, M.A., M.D., M.Ch.

The short paper to which I invite your attention this evening is to be regarded as a mere fragment—a brief contribution to the surgery of the ureter. This latter is a subject which during the last few years has engaged the attention of surgeons; and, still, how few instances of operation for stone in the ureter have been placed upon record. I think you will agree with me that it is incumbent on every surgeon who meets with such cases in his practice to publish details thereof, so that, in time, we may have accumulated a sufficiently large and varied number, as a basis from which to draw authoritative conclusions as to the best methods of dealing with stone impacted in any particular part of the ureteral canal. It is in this spirit that I venture to place before you details of two cases of operation for impacted ureteral calculus which have recently fallen to my lot, each in its way, I venture to think, possessing some features of interest.

Lieut. D., Royal Artillery, age 23, sent by Dr. Blood, of Woolwich, consulted me on July 19th, 1898. He had suffered, off and on, for 19 months from renal colic and hæmaturia. The symptoms first set in at Malta in January, 1897, in the form of colicy pains in both loins, extending across the abdomen, whenever he rode on horseback. These were at first considered to be due to indigestion; but on the third or fourth day, on dismounting, he found that his urine contained blood, so he went on the sick list. The symptoms passed off in a few days with rest and treatment

Three weeks later, his duties being heavy and involving much riding, the symptoms returned, and he had again to go on the sick list. The pain at this period was mainly confined to the left side, commencing in the loin, shooting down to the groin, and sometimes across the abdomen, giving rise to the sensation of a string being tied tightly round the body. At no period did the pain shoot down into the testicle, nor was the organ retracted.

The symptoms continuing, he came to England in May, 1897, and placed himself under the care of a well-known London physician. The renal attacks with hæmaturia continued to come on periodically every fortnight or so, and, strange to say, two of his worst attacks seemed to have been brought on by short sailing trips, though he did not suffer from sea-sickness. Getting no better he returned to Malta in August, 1897, and during the voyage painless hæmaturia set in, which continued, off and on, till September, when another severe attack of pain in the left loin occurred, and the hæmaturia suddenly ceased. He was then seen by Major Swabey, R.A.M.C., who diagnosed the case as one of stone in the kidney. Unable to continue at duty he went to Sicily, but the voyage again brought on hæmaturia, accompanied by fever, which, after lasting a week, again wound up with severe colic. He returned to duty at Malta at the end of the month of October, but had to lie down for several hours in the middle of the day to relieve the pain. The re-drilling commenced on November 1st, and the first route-march brought on pain and hæmorrhage. He was excused marching and rode in a carriage, but though the pain was less the hæmorrhage continued. In December he passed two stones by the urethra, cylindrical in shape and like red sand-stone. The acute pain up to this was in the left side; and from that period till July, 1898, though there was occasional hæmaturia and the urine contained albumen, there was no severe pain, merely a dull aching in both loins.

In May, 1898, he returned to England for duty at Woolwich. On July 10th he took a lot of exercise, and next day an acute attack of pain set in, in the right side, which lasted 72 hours continuously, during which he was kept under the influence of morphia by Dr. Blood. The pain was excruciating, accompanied by restlessness, nausea, and vomiting, with constant desire to micturate, only a small quantity of high-coloured urine passing

each time. For two days after this attack the urine was clear, but after that became thick and bloody again.

I found that there was a slightly movable tumour in the region of the right kidney as large as two fists, extremely tender on palpation both in the loin and in front. There was no stabbing pain on pressure at the back, but distinct tenderness at one spot in front, midway between the navel and the anterior superior spine of the ilium. Urine, colour of Madeira wine, acid, sp. gr. 1015, containing blood corpuscles and crystals of oxalate of lime, also albumen. The left kidney was normal. My diagnosis was—stone in the pelvis of the kidney, trying to pass down the ureter and causing obstruction of urine.

On July 30th the patient entered a surgical home, and on August 1st I operated, Mr. W. Braine giving the anæsthetic, and Mr. B. Smeaton assisting. Drs. A. M. MacDonnell and H. T. Griffiths were also present. The kidney was rapidly exposed through an oblique lumbar incision 4½ inches long, and the fatty capsule opened. No stone was detected on feeling it all over. The kidney, which was much enlarged and congested, was stripped of its fatty capsule and brought out on to the loin through the wound. I felt it carefully all over, but could detect no stone. The kidney was then opened through the convex border by means of a scalpel, and my finger passed into the pelvis, a rush of urine taking place through the wound, and the kidney collapsing much in size. No stone could be found, though my finger passed easily into the calyces, which, with the pelvis, formed a large smooth-walled cavity. I then passed a catheter down the ureter as far as 4 inches from the pelvis, where it was obstructed. A long silver probe was then passed, and a stone felt in the ureter at this point.

The incision in the loin was at once extended downwards and inwards for 4 or 5 inches, and the peritoneum raised inwards from off the ureter by my fingers, till I could grasp the stone (which felt the size of a filbert), lying in the ureter between my finger and thumb. I made several attempts to push the stone upwards into the kidney, but it was too firmly impacted in the ureter, so the ureter was incised longitudinally over the stone, which was pushed through the wound by the point of my finger. A bougie was then passed into the bladder through the ureter from the pelvis. I did not suture the wound in the ureter because (1)

suturing of the ureter when a stone is removed extra-peritoneally is a debatable proceeding; (2) the ureter was so narrow that suturing would probably cause contraction of the canal; and (3) though I could extract the stone with the limited incision already indicated, the successful suturing of the ureter at the bottom of a deep wound was impossible without a much more extended dissection. I contented myself, therefore, by placing a large drainage tube down to the opening in the ureter, along the back of the kidney, bringing the muscles together by buried silk sutures, and the superficial parts by silk-worm gut. The wound in the kidney was partially closed by two sutures, the middle portion being left open so that the urine might flow freely through the loin, and thus avoid tension on the ureter till the wound in it should have healed.

Bloody urine was passed freely by the urethra in the evening, showing that the ureter was patent; and bloody urine by both loin and urethra for several days. On the third day I began to shorten the drainage tube daily, and this was completely removed on August 7th. On the 8th the wound was firmly united, save in the track of the drainage tube. On the 9th urine ceased to flow by the loin.

The patient made a rapid recovery; was sitting up on August 28th; went for a walk on September 2nd; and left the home on September 7th, travelling by train to Margate. Soon after he began to bicycle; he started to rejoin his battery at Gibraltar on November 15th. He is now in excellent health, and wrote to me in the end of December last that he does five or six hours' work daily with his battery, ascending the Rock 1,300 feet.

It may be of interest to mention, as bearing on the hereditary tendency of stone, that owing to the successful result in this case, the patient's mother, who for 17 years had suffered from stone in the kidney, placed herself in my hands. On September 28th, 1898, I performed a lumbar nephro-lithotomy. Dr. Dudley Buxton, anæsthetist, and Mr. Swinford Edwards assisted me, removing one largish oxalate of limestone from the pelvis, and several smaller ones from the calyces. The kidney, which was extremely large, was sutured. No urine passed by the wound, and the patient made a rapid and successful recovery. Furthermore, this lady's father suffered severely from gravel for many years before his death. Some years ago I operated successfully

for stone in the bladder on three generations in the same year—son, father, and grandfather.

The second case, a man, aged 53, has been a patient at St. Peter's Hospital, off and on, for 13 years, suffering from multiple stricture of the urethra, which had been operated on several times, and symptoms of kidney stone. In 1895 his left kidney was explored for stone by another surgeon, but with a negative result. Temporary relief ensued, but the old symptoms again returned, and in the summer of 1897 he was very ill, the pain in the left loin being very severe at times, radiating to the groin and testicle, and accompanied by hæmaturia, which at times was very profuse; there was also occasionally pain in the right loin. The urine was extremely thick and offensive, and on standing deposited pus amounting to half its bulk. I determined under these circumstances to explore the left kidney again, which was accomplished on September 1st, 1897, by an oblique lumbar incision. A most careful search was made, but no kidney was found, though I passed my fingers well up under the ribs, down to the iliac fossa, and inwards to the aorta, pushing the peritoneum out of the way. The patient rapidly recovered from the operation, but there was only a very temporary relief to the symptoms, which soon set in as bad as ever. Under the impression that the symptoms might in large part be due to backward pressure, resulting from the stricture, which had again contracted, so that a No. 6 bougie, English scale, was passed with difficulty, I performed internal urethrotomy on November 18th, 1897, cutting three dense cartilaginous strictures up to 16 of the English scale. The patient was extremely ill after the operation for two days with severe rigors, the temperature rising to 106° F., and the urine being very scanty; but he recovered, though slowly, leaving the hospital early in January. This operation, though it afforded a free flow for the passage of his urine, did not much diminish his other symptoms. Though the pain in the left loin gradually diminished, that in the right loin increased, and for months he continued to attend the out-patient department, his urine constantly containing much pus, and at times a considerable quantity of blood. Occasionally the right loin became much swollen, and the disappearance of the swelling was always followed by an increase in the quantity of pus in the urine. In October, 1898, he began to complain of great irritability of the bladder; there

was constant desire to pass water with pain at the end of the penis. He was admitted into hospital, and on November 9th I cystoscoped him under an anæsthetic, and detected a long, narrow, rough, pencil-shaped stone projecting into the bladder from the right ureteral opening. The portion of stone projecting into the bladder appeared by the cystoscope to be about three-quarters of an inch long, and dark grey in colour. My colleague, Mr. Reginald Harrison, and others had an excellent view of the stone. I at once introduced a lithotrite, and after three or four unsuccessful attempts, eventually caught the stone between the blades, pulled it out of the ureteral opening, crushed it, and removed the *débris* by the aspirator. The *débris* consisted of urates and weighed 12 grains. On November 23rd the patient was again cystoscoped. The bladder walls were coated with mucus; both ureters were seen to be acting, and flakes of pus were seen issuing from them.

The patient has continued to improve steadily in health. The pains in the loins have disappeared; there is no hæmaturia and scarcely any pus in his urine. He states that he is in better health than he has been for years.

The history of this case presents many points for speculation which would, however, be foreign to the purpose of this paper; the case being now brought forward only for the purpose of illustrating what I believe to be a novel method by which a stone projecting into the bladder from the ureter may possibly sometimes be successfully removed.

I will, in conclusion, briefly refer to five other instances of stone impacted or encysted in the ureter, which have been operated on by me—four of these during my experience in India. In three, the stone was impacted at the ureteral orifice, projecting into the bladder and felt by the sound. One occurred in a female, two in males. In each instance an operation was undertaken, under the impression that I had to deal with an ordinary case of stone in the bladder, and in each case the stone was dislodged from its position by introducing the forefinger through the dilated urethra in the female and through the ordinary perineal lithotomy wound in the males, scraping the orifice of the ureter by the finger nail, the stone being forced downwards and inwards by the hand placed on the groin, and then removed by the ordinary lithotomy forceps.

In the 'British Medical Journal,' May 9th, 1891, p. 1005, I have given details of a boy, aged 15, from whom I removed, at two different sittings, by litholopaxy two separate stones, weighing respectively 757 and 581 grains, aggregating more than 3¼ ounces. After removing the first stone from the bladder I found that there was a second lying in the lower part of the right ureter close to the bladder. The second stone was felt as a hard tumour in the loin, about 3 inches long, and could easily be felt in the ureter between a finger in the rectum and the hand on the groin. I determined to remove this stone by suprapubic cystotomy as soon as the patient should have recovered from the first operation, but on the fourth day the tumour in the groin suddenly disappeared, and a stone was felt lying free in the bladder, and successfully removed by litholopaxy. Evidently this latter stone, lying in the ureter, rested on the stone located in the bladder; as soon as this support was removed the ureteral calculus was pushed into the bladder by the force of the accumulated urine behind it.

The fifth case is recorded in the 'Medico-Chirurgical Transactions,' vol. 81. It was that of an obscure cystic tumour of the bladder, which contained two calculi, weighing 41 grains, removed suprapubically. My explanation of the case was this—that the stones descending from the kidney were arrested at that portion of the ureter which passes obliquely through the bladder wall, and that, unable to escape owing to a contracted orifice, they bulged the bladder wall inwards, forming a smooth cystic tumour which was recognised by the cystoscope, and successfully dealt with in the way indicated.

Mr. Bruce Clarke referred to the case of a woman with symptoms referred to the right kidney, which he first regarded as due to stone or to floating kidney. She had suffered for about two years. He made the usual lumbar incision, and found the kidney larger than normal. He then made an incision into the kidney at the junction of the pelvis with the kidney substance, introduced his finger, and, with a short sound, explored the whole of the interior without finding anything. He next explored the ureter with a bougie, which was arrested about 5 inches below the kidney. He insinuated his finger some distance down outside the ureter till he could feel the exact spot, after which he lengthened the incision, pushed up the peritoneum, and found a stone firmly impacted; he made an incision into the ureter and removed it. He did not suture this, because it was practically impossible, but brought out a drainage tube in the loin and also one from the kidney. The patient made an excellent recovery, and he had since seen urine issuing from both

ureters with the cystoscope. Stones which were impacted in the lower part of the ureter probably accounted for the condition, occasionally met with, of encysted calculi, at or about the orifices of the ureter. If the stone got fixed just at the orifice of the bladder, it might ulcerate through, and then one got a pouch in the bladder wall which contained a stone, and was in communication with the ureter. He had seen one such case during life, and several on the *post-mortem* table.

The Vice-President observed that everything relating to stone in the ureter was of interest to all of them. Commenting on the fact that a stone escaping from the pelvis of the kidney was arrested either at the commencement of the ureter or at its entrance into the bladder, he asked what were the author's views as to the exact situation at which the stone was usually arrested between these two points?

Mr. Freyer, in reply, said it was a general impression that a stone was arrested either at a point about 2 inches from the pelvis of the kidney or else quite close to the bladder; but they had now a sufficiently large number of cases on record to show that the stone might be arrested at almost any spot in the ureter. This impression had arisen from the fact that stones in any other than these two positions could not possibly be found without an elaborate dissection. If below, it could sometimes be felt by the sound, seen by the cystoscope, or, in the female, felt by a finger introduced through the dilated urethra. A stone anywhere else would not be detected. In most cases the operation was undertaken under the impression that there was a stone in the kidney or in the pelvis of the kidney, and it was only when no stone was found in these two situations that the ureter was explored, often with a successful result.

February 6th, 20th, and March 6th, 1899.

LETTSOMIAN LECTURES: SOME CLINICAL ASPECTS OF GRANULAR KIDNEY.

By Samuel West, M.D., F.R.C.P.

LECTURE I.

The invitation to deliver the Lettsomian Lectures before this ancient Society has been accepted in times past with full appreciation of the honour by many of the most distinguished members of the profession. The great names upon that list have aroused in me the natural fear that my ability may prove unequal to the task, yet I trust that with their example and your indulgence I may succeed in making the present Lectures not unworthy altogether of a place in the series.

In choosing granular kidney, I was guided by the wish to select a subject which should be of practical importance, and therefore of general interest, and at the same time one upon which I could speak from personal experience.

Granular kidney is a disease of great importance on account of its frequency, a frequency which is by no means adequately recognised. It is often discovered *post-mortem* when not suspected. It is often in itself the cause of death, even of sudden death. It often explains why death has happened in other diseases which otherwise might not have proved fatal. During life it is often discovered unexpectedly if looked for; it is often overlooked if not suspected, and it often explains a case which has been a puzzle until granular kidney gave the key to its solution.

Its Frequency and Importance.

The general *post-mortem* frequency of granular kidney ranges from 11·8 to 18 per cent., so that it is a very common condition.

Thus the *post-mortem* records at St. Bartholomew's Hospital for a period of five years gives 13·2 per cent. (219 cases of granular kidney in 1,662 necropsies, children being excluded from the calculation).* Of these, 132 were in males and 84 in females. Goodhart's statistics at Guy's yield 15 per cent.; Saundby's, at the Birmingham General Hospital, 18 per cent.

Mahomed's Table showing Relative Frequency of Granular Kidneys to Healthy Kidneys at different Age-Periods based on 336 *Cases, of which* 57 *were above* 50.

Ages.	Granular Kidney found in
10–20	2·3 per cent.
30	3·0 „
40	12·0 „
50	38·5 „
60	43·0 „
Above 60	50·0 „

In fatal cases of cerebral hæmorrhage Dickinson found granular kidney in no fewer than 41 per cent. In the St. Bartholomew's Hospital figures referred to the percentage was 21·5 (34 cases in 158).

* For these figures I am indebted to Mr. Woodbridge. They will be published *in extenso* in the 'St. Bartholomew's Hospital Reports.'

As a cause of sudden death nothing will better prove the importance of granular kidney than the following statistics.

They were obtained for me by Dr. Horder,* as the result of an investigation made into the cause of death in persons brought into St. Bartholomew's Hospital dead or dying. The total number of such cases (excluding children under 5) was 79, in all of which a *post-mortem* examination was made. Of these, 64 were dead on reaching the hospital, and 15 died in the surgery.

Table showing the frequency with which Chronic Interstitial Nephritis was found in the Bodies of Persons brought in Dead or Dying into St. Bartholomew's Hospital during the period of five years (1894–8 *inclusive*), *Children under* 5 *excluded.*

Chronic Interstitial Nephritis found to be	64 cases brought in dead.	15 cases dying in surgery.	79 cases together.
1. The actual cause of death in	13 cases = 20·3%	2 cases = 13·3%	16·8%
2. A concomitant cause of death in	19 cases = 30%	2 cases = 13·3%	21·6%
3. An accessory but doubtful cause of death in ..	6 cases = 6·25%	2 cases = 13·3%	9·77%
Totals	38 cases = 56·25%	6 cases = 40%	48·17%

In 48 per cent. of these cases chronic interstitial nephritis was present. In 16·8 per cent. it was the only cause of death, and in 21·6 per cent. more it played its part either in producing death or in causing the lesion which led to death—that is, together in 38·4 per cent.

Nothing more is necessary to prove the importance and frequency of the disease.

Of the 21 cases in which granular kidney was the concomitant cause of death, in 13 death was due to hæmorrhage, to which the interstitial nephritis directly or indirectly led.

Of these, in one there was hæmorrhage into the pons varolii; in one pachymeningitis hæmorrhagica; and the remaining case was one of hæmoptysis.

In four cases aneurysm of the aorta was present, the rupture of which was the cause of death; and in the remaining three death resulted from the rupture of an aneurysm of the anterior communicating artery in one instance, and of the sylvian artery in two more.

* This paper will be published *in extenso* in the 'St. Bartholomew's Hospital Reports' for 1899. The short abstract now given of it is of the nature of a preliminary report only.

The association of interstitial nephritis and aneurysm, both of the aorta and of other arteries, is remarkable, and, so far as I know, has not been clearly shown before.

In seven cases the cause of death was cardiac.

Acute endocarditis was found in	1 case.
Acute endocarditis with pericarditis in	1 „
Aortic incompetence in	3 cases.
Aneurysm of the heart in	1 case.
Failure of heart associated with atheroma of the coronary artery in	1 „
	7 cases.

Of the remaining cases three had phthisis and one pneumonia.

The influence of granular kidney upon the prognosis of other diseases is well illustrated by acute pneumonia.

The association of pneumonia with any pre-existing disease greatly increases its gravity, even, according to Huss, to the extent of making it four times as fatal as in uncomplicated cases. In 100 fatal cases which I investigated, 23 showed some pre-existent chronic disease of the lungs or pleura, eight of the heart, and 10 of the kidneys.

Sturge's percentage was somewhat higher, namely, 15 per cent.

For all the reasons given, granular kidney is not only one of the most interesting of diseases, but also one of the most important.

Lesions.—Physical Signs.—Symptoms.

The lesions, physical signs, and symptoms of granular kidney all alike fall into two groups, the cardiovascular and the renal.

The physical signs are early, the symptoms late. When symptoms arise the disease has already existed for some time, and the lesions are far advanced. For the diagnosis of the disease in its early stages we must look to the physical signs and not to the symptoms.

The Lesions.—Morbid Anatomy.

Granular kidney is a bilateral and to a great extent symmetrical affection, that is to say, both kidneys are similarly affected, though not necessarily to the same extent. The difference between them however, if there be any, is slight, and rarely exceeds an ounce.

The kidneys may be much contracted and greatly reduced in size, so that they may not weigh more than $1\frac{1}{2}$ or 2 ounces each.

Extreme contraction such as this is really rare. The kidneys are, it is true, usually reduced in size and weight, but by no means to the extent that seems generally believed; while in some instances, so far from being smaller than normal, they may be much above the average size and weight. In other words, there are large as well as small granular kidneys, just as there are large and small cirrhotic livers.

I have taken the weights of 140 cases, and put them in a diagrammatic form, in which the weights are indicated by the vertical height and the number of cases by the divisions on the base line.

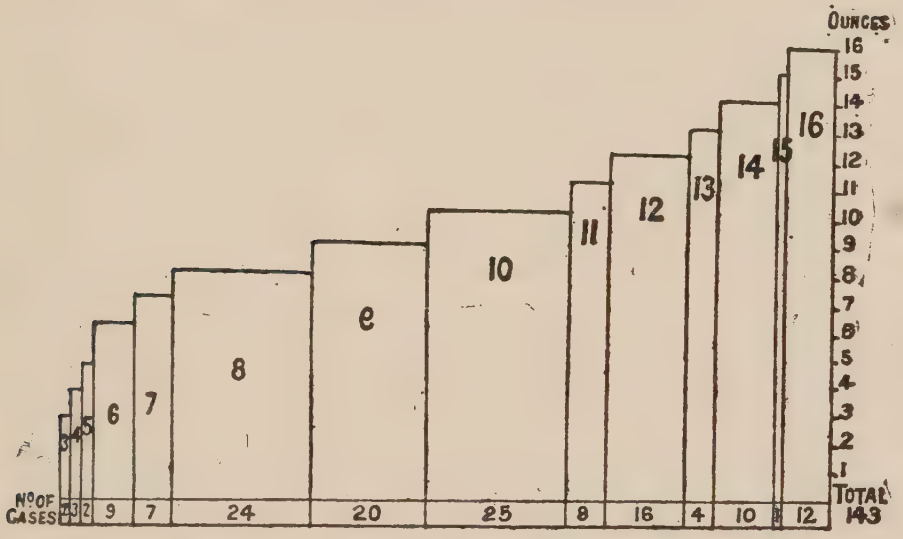


Diagram in which the weights in ounces of the two kidneys together are shown by the height of the vertical line, while the number of cases in which the kidneys were of the given weight is shown by the length of the base line.

Taking 11 ounces as the mean weight of the two kidneys together in the two sexes, or even including the cases in which the weights reached 12 ounces, it will be seen that in the great majority of cases the kidneys ranged between 8 and 10 ounces, and that in only 5 per cent. was the combined weight less than 6 ounces. On the other hand, in no fewer than 20 per cent. the combined weight was above the normal average, and in 12 cases reached as much as 16 ounces. In a few of these, no doubt, some allowance ought to be made for the fat deposited round the pelvis of the kidney, and in some others the weight was partly to be explained by the number and size of the cysts present. But in the majority neither cysts nor fat took any material part in the increase of weight, and in two instances in which the kidneys weighed 16 and 12 ounces respectively they were described in the *post-mortem* report as "large granular kidneys."

The kidneys, moreover, are not always granular on the surface, though microscopically the interstitial change may be marked enough.

It is also stated that the kidneys may be small and granular in appearance, and yet on section show no interstitial change. Such cases have been mentioned to me, but I cannot say that I have ever met with them myself.

As granular kidneys are not always granular, it might be thought some more accurate term could be found for its description, such as cirrhosis of the kidneys; but granular kidney is a term so well known and so generally accepted that it would be difficult to substitute for it any other which, though more accurate, would be likely to be adopted.

The typical granular kidney is small, contracted, hard, with a granular surface and often numerous cysts. On section the whole kidney is found to be cirrhotic and wasted, but the wasting affects chiefly the cortical region.

The changes consist in fibroid induration and cellular degeneration.

Though often described as chronic interstitial nephritis, it is an open question how far these changes are in reality of inflammatory origin. Certainly in most cases we have no more evidence of inflammatory processes in the cirrhotic kidney than we have in the cirrhotic liver.

Forms and Origin.

White and Red.—Granular kidneys are sometimes described as of two kinds, the WHITE and the RED, and the difference in colour is held by many to denote difference in origin, and they are accordingly called the *contracted white* and the *contracted red.* The small white is generally stated to be somewhat larger than the red, to be less granular on the surface, and to have more cells left, these cells being in a condition of fatty change, to which change the colour is referred.

None of these statements are, in my experience, absolutely correct; for small white kidneys are often quite as small and as granular on the surface as the small red, and may have quite as few or even fewer cells left, and these cells are often not fatty.

The term "contracted white" introduces confusion, and seems to assume what ought to be proved—namely, the different origin of the two forms. It suggests that the small white is but the later stage of the large white kidney. It assumes that every stage

can be demonstrated between the large white kidney of chronic parenchymatous nephritis, through the contract*ing* white to the contract*ed* white.

If this were the usual sequence of events, it ought to be capable of easy clinical proof. Pathologically it is easy to show that chronic parenchymatous or the large white kidney presents interstitial changes of a contracting kind, but the weak link in the chain is between the so-called contracting form and the contracted form.

The two forms certainly exist, but if the distinction between the two forms is to be made it would be very much better to speak of them as small white and small red, or as white cirrhotic and red cirrhotic, rather than white and red contracted kidneys. For myself, I consider the difference is really one of colour chiefly. Microscopically I do not see that any clear distinction can be drawn. The colour does not depend entirely upon the condition of the cells as is usually stated, for the fatty change to which the white colour is referred is not constant, and such kidneys may be markedly white or pale, in which microscopical examination shows a remarkable absence of cells, while the cells that remain are not so much fatty as granular. If pathologically the distinction between the two forms is open to question, clinically, so far as I can see, no distinction at all can be drawn between them; that is to say, with exactly the same clinical symptoms we may find the kidneys in the one case white and in the other case red.

It seems to be sometimes assumed that the small white and small red are but different stages or degrees, that is, that the white kidney would in time become red, but even that is not true. On the contrary, it might with equal justice be argued that the pale cirrhotic kidney is in some cases an accidental condition of the red, and that a red kidney would become pale if the cells became inflamed and degenerated.

If this did prove to be the case it might explain why in the course of granular kidney acute symptoms so often develop suddenly as they do, and lead rapidly to death—the few active gland cells left in the wasted kidney becoming suddenly inflamed and degenerate, with the result that the kidneys cease to do their work, and life is brought rapidly to an end.

We might thus possibly connect the sudden onset of acute uræmia, *i.e.*, acute renal toxæmia, with the condition of the renal

cells, just as in cirrhosis of the liver we associate the sudden onset of acute hepatic toxæmia with acute degeneration of the liver cells.

Granular kidney is often described as chronic interstitial nephritis, but all forms of chronic interstitial nephritis are not necessarily granular kidney.

First of all must be cut out of the category of granular kidney all those cases in which the lesion is unilateral, for example that which results from obstruction to the ureters.

The first effect of obstruction to the ureters is felt by the cells in the kidney, which lose their vitality, and ultimately atrophy and disappear. The ultimate results may be two—either fluid continues to be secreted and a condition of hydronephrosis is set up, or the fluid ceases to be excreted and the kidney shrinks. In the latter case fibrotic change occurs, and in the end the kidney may become greatly contracted and typically granular. Extreme changes of this kind consequent on obstruction, can, of course, only occur on one side.

Again, all cases of patchy fibrosis—for example, the results of infarcts or gummata—even if both kidneys are involved, must be excluded, for they do not produce the clinical symptoms of granular kidney.

Even when the lesions are bilateral, and both kidneys are affected, I should still exclude from the category of granular kidney certain forms of chronic interstitial nephritis, which are often included pathologically, though clinically they are distinct. I refer to the degenerate kidneys met with in connection with advanced atheromatous disease, or with the chronic gout of elderly persons; in fact, the kidneys which used to be commonly called senile or gouty. These kidneys are often markedly interstitial, and there is a good deal of fatty and cellular degeneration, giving the surface a mottled appearance, but they need not necessarily be granular, and are usually large. Moreover, the case need not, and indeed does not generally, run the clinical course of granular kidney.

Eliminating these various forms we are left with a pathological group of cases of interstitial nephritis of definite character, which we commonly recognise by the term granular or cirrhotic kidneys.

Cause.

Granular kidney being a bilateral affection must ultimately depend upon some general cause, acting no doubt through the

intermediation of the blood, for there is nothing else except the nerves which could explain the symmetry of the disease.

Whatever the ultimate cause, the fibrotic changes in the kidney might arise in connection with some primary affection of the gland cells or some primary affection of the non-glandular parts; for example, the connective tissue itself or the structures contained in it, namely, the arteries, veins, lymphatics, or ureters.

Of primary affections of the connective tissue itself nothing is known, nor is anything known of primary affections of the lymphatics of the kidneys, though this much is known, that they are secondarily involved in all forms of inflammation, whether acute or chronic. We have no evidence that venous obstruction can produce cirrhosis in the kidney any more than it does in the liver. In considering, therefore, the causes of granular kidney, we have only the arteries and cells left as the primary sources of the disease.

In most recent writings granular kidney is divided into two forms, the *arterio-sclerotic* and the *renal*. These are often dealt with in different places, the one form under diseases of the vessels, the other under diseases of the kidney, so that in order to gain a complete view of granular kidney as a disease it has to be read in different chapters. This is very inconvenient and confusing, and suggests a marked difference between the two forms which, clinically, does not exist. It has, however, one advantage in that it fixes attention upon the question which is still at issue, namely, whether it is in the vessels or in the kidney itself that the primary causes of the disease are to be sought.

I shall now consider, as briefly as possible, the relation in which granular kidney appears to stand, on the one hand to inflammatory affections of the kidney, and on the other to the degenerative disease of the vessels.

The Relation of Granular Kidney to Acute Nephritis.

Acute nephritis attacks the cells primarily and chiefly, but the interstitial tissues are always involved to some extent, and the more so the longer the disease has existed. It is by the lymphatics that the products of inflammation to a great extent are removed,

and in passing they appear to set up some irritation, so that in course of time a small-celled infiltration is found, in parts at any rate, following the lymphatics and surrounding the blood vessels. This small-celled infiltration may in time end in the production of connective tissue, at first in parts only, in other cases more widely distributed, and there will be the more of it the longer the disease has lasted.

I do not know that the distinction which is often made between the different forms of acute nephritis, according as it attacks the glomeruli or the convoluted tubes (for instance, acute diffuse nephritis and glomerular nephritis), is one which is borne out by observation. In most cases all parts of the kidney are affected, though one may be affected more than another. Whether the distinction be just or not, I do not think it bears in any important way upon the question as to the relation between acute nephritis and granular kidney.

The large red kidney of acute nephritis passes, as the disease lasts, gradually into the large white. In the large white kidney some interstitial change is often found, sometimes local, at other times more diffuse; and, admitting that pathological connective tissue contracts in the kidney as elsewhere, we should expect the large white kidney in time to become smaller, to undergo a certain degree of contraction in parts, if not as a whole, and of this we have frequent pathological proof. The kidney is then described as *contracting white.*

It is even conceivable, theoretically, that the contraction might go much further, so that in time we might have the large or *contracting white* kidney passing into what may be described as a small, or *contracted, white* kidney, or even possibly ending in a small *contracted red* kidney.

Now, admitting the pathological possibility that acute parenchymatous nephritis might end in a granular kidney, if this actually occurred frequently it ought to be capable of easy clinical proof, for the symptoms of acute nephritis are not such as are likely to be often overlooked, by the patient or by the friends. Such cases are recorded, but they are certainly rare, and many of them prove, on careful critical investigation, not to be so conclusive as they at first sight appear. I have been on the watch for them for years, and although I have seen many in which the lesion has gone so far as to produce a somewhat diminished white kidney,

the majority of such kidneys are still much above normal size. I have never traced a single case beyond this—that is, as far as a small white kidney. I do not say that it never can be so traced; on the contrary, I believe it may. I merely say that, so far, I have failed to observe it. On this point the late Professor Kanthack told me that his experience was the same, and that pathologically he had not been able to satisfy himself that an acute parenchymatous nephritis did actually pass beyond the contracting white stage.

Appended to these lectures are two cases in which the disease was traced from the commencement of acute nephritis to death after some months in each case, and in which microscopical examination revealed more or less extensive fibrosis in the kidneys, though they were in neither case granular.

Macpherson's case (female, aged 29) seemed to be one of ordinary chronic parenchymatous nephritis, and so the kidneys appeared *post-mortem*. I had the kidneys carefully examined with the microscope, because shortly before death, which occurred after about nine months' illness, albuminuric retinitis had been found. The ordinary lesions of chronic parenchymatous nephritis were found, but with them also scattered patches of cortical fibrosis.

Blakeborough's case (female, aged 14) is a more remarkable one, for on the child's death, after about 12 months' illness, the kidneys, though of large size and not granular, and presenting the appearance of large white kidney, showed the most remarkable fibrosis, which did not involve the cortex alone, but the medulla too, and every part of the kidney uniformly. In this case, also, albuminuric retinitis was discovered about four months before death.

It might be urged that marked contraction in granular kidney would be more frequent if the patients lived long enough, but that they die too soon, perhaps after but a few months' illness, and before the interstitial changes have had time fully to develop. This is but to acknowledge that it does not necessarily or often occur.

But it does not necessarily follow, even when the symptoms of chronic parenchymatous nephritis have lasted for many years, that a contracted kidney will result.

I have had under my care a woman, who has had several attacks of parenchymatous nephritis in the course of 13 years, and who has never lost the albumen from the urine; yet now, at the end of 13 years, her arteries are thin, and her heart not hypertrophied; she appears in fair health, and her retinæ are normal; there are, in fact, in spite of the long

duration of the case, no clinical signs whatever which would justify the diagnosis of granular kidney.

I know also of another case in a man who, 25 years ago, had parenchymatous nephritis of great severity, and who has continued to pass albumen in large amounts (one-third to one-half) ever since, and yet no signs of contracted kidney have so far shown themselves. Cases of shorter duration than these are not uncommon.

Nor can any closer relation be proved by the clinical history to exist between granular kidney and antecedent acute nephritis. It is quite unusual in cases of granular kidney to obtain a history of symptoms which would in any way justify the diagnosis of acute nephritis. This fact has been always insisted on as long as granular kidney has been recognised as a disease, and is generally accepted.

Even when the history of antecedent acute nephritis is obtained, it does not necessarily follow that the granular kidney has been the result of it. The occurrence of acute nephritis is no proof in itself that prior to the acute nephritis the kidney was sound; or, in other words, that if after an attack of acute nephritis the kidneys are found granular, they became granular as the result of the acute attack. For, on the one hand, it is not uncommon, in the course of a case already recognised as one of granular kidney, to observe acute parenchymatous nephritis develop; and, on the other, to find in a patient, hitherto believed to be healthy, and presenting the symptoms of acute parenchymatous nephritis, thickened arteries and even eye-changes, which show that the disease really dated long before the commencement of the so-called acute attack—in other words, that the patient has got acute nephritis in both cases alike because the kidneys were already diseased. Indeed, I think we may almost go so far as to say that as in children acute parenchymatous nephritis raises a strong presumption in favour of a recent attack of scarlet fever, so in the adult it ought to suggest the suspicion that the kidneys were previously unsound—in other words, granular. Of this I am quite sure: that if, in cases of acute nephritis in the adult, the signs of granular kidney be looked for, they will very frequently be found.

In cases of granular kidney it is clear, therefore, that the history may be misleading, for an acute nephritis, if there be the history of an attack, may be the consequence of a pre-existent granular kidney quite as likely as the cause of it.

If, then—in spite of the fact that in the majority of cases of granular kidney the history and symptoms of nephritis are lacking—granular kidney is still to be referred to some antecedent nephritis, it follows that the initial attack must either have been slight, and therefore overlooked, or else that it was of some peculiar and specially latent kind.

Latent acute nephritis, diffuse or glomerular, in children, following diphtheria and other specific diseases, has been described and regarded by some writers as the germ of subsequent disease; but this is, of course, presumption and not evidence; it is opinion and not fact.

Acute nephritis certainly occurs sometimes without any obvious symptoms to suggest kidney disease.

As an illustration in point I may quote a case of a girl of about 10 years of age. I was asked to see her because she had been ailing in an indefinite way two or three weeks. The mother was alarmed when she noticed the urine to be dark in colour. The child showed nothing except an appearance of anæmia and debility, with a little dilatation of the heart in consequence, but on examining the urine it was found to contain blood and a large amount of albumen, and very numerous hyaline and epithelial casts. The child was treated in the usual way, and in two or three weeks recovered completely. The attack was ultimately traced to a sore throat, no doubt of a diphtheritic nature, which the child had suffered from some three weeks before, for one of the other children came under observation with paralysis of the muscles of accommodation in the eye about the same time.

The interest of this case lies in the fact that it shows how easily a fairly intense nephritis could be overlooked—as, indeed, it was overlooked—for the time.

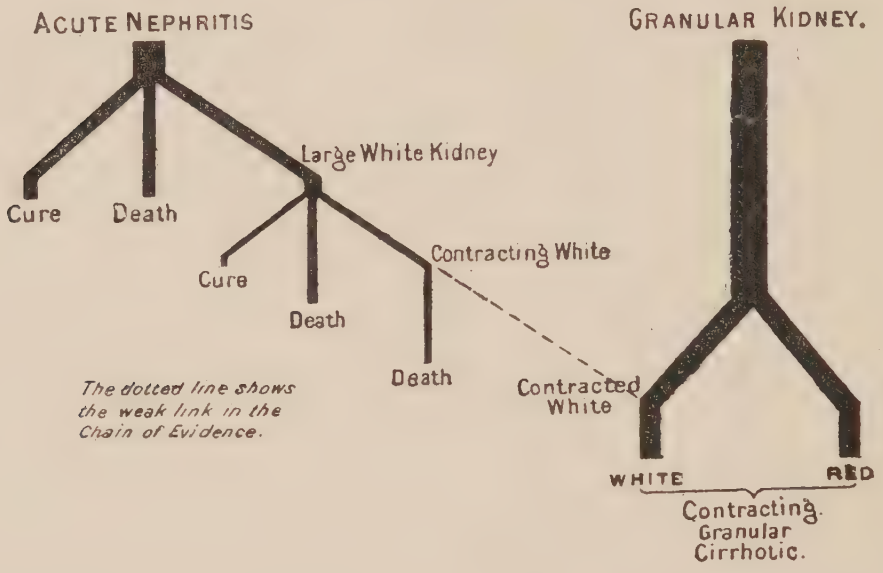
It is evident from what has been said that it is only in a very small number of cases of granular kidney that a history of antecedent acute nephritis can be obtained; that in still fewer instances can a case be traced from an acute initial acute nephritis to granular kidney.

It follows, therefore, that most cases of granular kidney must be referred to some other origin.

The diagram on next page shows the relation which is assumed to exist between the acute nephritis and the white cirrhotic kidney—the dotted line shows the weak link in the chain.

So far as the contracting white form the links are complete—the weak link lies between the *contracting* white and the *contracted* white, *i.e.*, the pale granular kidney.

The increased tendency to refer granular kidney to acute nephritis which is obvious in many writers of the present time is due, to a great extent, to the question being regarded too much from an anatomical, that is, pathological, instead of a clinical point of view. Pathologically, of course, such a sequence of changes is quite possible, but the theories of pathology must be checked and corrected by the facts of clinical observation. The theory is one which ought to be capable of easy clinical proof, but the clinical proof, as far as I can judge, is lacking.



It would not have been necessary to discuss so fully the want of relation between granular kidney and acute nephritis had it not been for the frequent assumption of this connection at the present time by the sharp distinction so often drawn between the two forms of granular kidney, the white and the red.

The Relation of Granular Kidney to Arterial Disease.

The changes in the arteries with granular kidney are general, widespread, in fact universal, throughout the whole body. They are found in all parts alike—in the kidney, for instance, as well as in the brain, eye, spinal cord, or even skin.

There are only two forms of general arterial change recognised in pathology, namely, atheroma and that connected with granular kidney.

Atheroma is a well-marked disease of the vessels. It is the degeneration which is usual as age advances, and though sometimes met with in earlier life, and even sometimes in quite young people, it is certainly a comparatively rare affection at the time when the changes of granular kidney are most common. Moreover, the thickening is characteristic; it is irregular and not uniform, and, when well-marked, can easily be diagnosed by the finger in superficial arteries like the radial and temporal.

Atheroma being a general disease will, of course, affect the renal arteries like any other, and will produce in the kidneys similar changes to those to which it leads in other cellular organs—for example, in the brain. Thus the obstruction to the circulation produced by the arterial changes will lead to malnutrition and fatty changes in the cells, and these may ultimately atrophy, and as the result of this a certain amount of connective tissue may be formed in the interstitial tissue of the kidney. Thus it is not at all uncommon in the later periods of life to find the kidneys large and mottled, showing a good deal of interstitial tissue and of cellular degeneration; yet these atheromatous or senile changes do not as a rule, though they might conceivably, lead to the well-known granular contracted kidney.

The difficulty is further increased by the fact that the atheromatous changes in the arteries, if widespread, lead to secondary changes in the heart. Hypertrophy and subsequent affections of the heart follow, which so far resemble those met with in granular kidney.

Of course, old persons may suffer from granular kidney, and the two conditions may be associated. But for all that, if the question be regarded without bias, and in a general comprehensive way, it must be acknowledged that atheromatous disease of the arteries and the changes of granular kidney are different in kind as well as in history and clinical course.

In *granular kidney* the thickening of the vessels is fairly uniform, at any rate it does not occur in the irregular patches of atheroma, and is universal.

As to the nature of the vascular lesions dispute has long raged, the one side maintaining that it was of the nature of hypertrophy, the other of a degeneration. The facts are that both changes

are found, the hypertrophic changes in the earlier stages and the degenerative in the later.

It is the changes in the arteries that are of prime importance in granular kidney, for in great measure, if not entirely, the hypertrophy of the heart is secondary to, and the consequence of, the changes in the vessels. Hypertrophy of the heart is met with, of course, where the vessels are atheromatous, as well as where they are the subject of the changes peculiar to granular kidney. It is unfortunate that in many of the investigations as to the frequency of hypertrophy of the heart in renal diseases more accurate note has not been taken of the general condition of the vessels for the purposes of excluding atheroma, for this omission deprives the results of much of their value, and at the same time probably explains their divergence.

The Relation between the Cardio-Vascular and the Renal Lesions.

So far as the cardio-vascular symptoms go I think we may assume that the cardiac lesion being of the nature of hypertrophy is, as in other cases, the response of the heart to some extra work thrown upon it of a permanent kind; it must therefore be secondary.

The hypertrophy of the arteries also is probably, like that of the heart, secondary; for we cannot suppose that there is any antagonism between the heart and the arteries, though this was once assumed to be the case—the heart hypertrophying, it was then supposed, in order to overcome the obstruction offered by the hypertrophy of the vessels. It certainly seems more probable that both the heart and the vessels hypertrophy together for the purpose of assisting the circulation to overcome some obstruction.

If this be so, the obstruction must be peripherally seated, and must be sought in the small peripheral arteries or capillaries. In these peripheral vessels the possible causes of obstruction are two: either there must be a *structural change*—that is to say, an anatomical lesion, or some *functional disturbance* which interferes with the passage of the blood through the vessels.

(1) If the change be structural, and the lesions of the peripheral vessels are of a degenerative character, being general throughout the whole of the body, they would involve the kidneys as well

as other parts, and in this way it is conceivable that the kidney disease might be a co-ordinate or subordinate part of the general vascular disease. This is a theory which is now indicated by the term arterial sclerosis, and such kidneys are described as arterio-sclerotic kidneys.

(2) If, on the other hand, the change be not of a structural kind, but a functional one, any structural change that is found being of a secondary and subsequent order, the obstruction must depend upon some impurity in the blood.

But this impurity of the blood, which renders it more or less obnoxious to the tissues, may be either of renal or extrarenal origin. If of extrarenal origin then the result may be the same as in the former case—namely, a general vascular disease which affects the kidney as well as other parts. But it may also be of renal origin, in which case there must have been some renal mischief antecedent.

Thus, even from the point of view of the vessels, we are brought back again to the two original views about which so much discussion has raged, namely, whether the disease is primarily arterial or primarily renal.

If the changes in the vessels be regarded as primary, as they certainly are in atheroma and may be also in granular kidney, then it might be possible to find cases of marked changes in the vessels with little or no changes in the kidney. And the same would be true if the changes in the vessels and in the kidney both stood in relation to some common cause.

It is only by studying the beginnings of the disease, that is to say, granular kidney in its early stages, that we can hope to arrive at a solution of these difficulties; for in the later stages all these changes are present together, and it is difficult, if not impossible, to form any sound opinion as to precedence of the one or the other.

If the renal changes were primary (and that whether the explanation be mechanical or pathological) we might expect to have some evidence of similar changes in the vessels as the result of other bilateral affections of the kidney, which are not of the nature of granular kidney—for example, where both kidneys are diseased, by obstruction to the ureters, or as the result of bilateral tuberculous disease, &c. Instances of the

kind are recorded, and even instances in which the general changes have been associated with unilateral disease of the kidney.

Making all allowance for exceptional cases, I think it must be admitted that, speaking generally, the vascular changes and the subsequent course of such cases are not those in granular kidney. Any given case which appears to break the rule requires to be carefully worked out, when it may be found after all to prove it.

If the changes in the vessels were primary then we ought to have evidence of vascular disease before we get evidence of kidney disease, and there ought to be cases in which, at the time of death, the kidneys are found not in a condition of granular change.

Cases of this latter kind are described—that is to say, cases in which the clinical features of granular kidney have been present during life, but at death, though the vessels were diseased, the kidneys were not found granular. Of course, two questions at once arise:—(1) Whether these were really instances of what we should clinically call granular kidney? and (2) Whether the kidneys, though not granular in appearance, were not fibrotic, as in the case I have previously referred to?

From whatever point of view, then, we regard granular kidney, whether pathological or clinical, we come to the same conclusion—namely, that whether primarily arterial or primarily renal granular kidney is a disease, *sui generis*, and ought on that account to be treated in writing on the subject under a separate heading, and not split up, as it generally is, between diseases of the arteries on the one hand and diseases of the kidneys on the other.

Analogy to Cirrhosis of Liver.

It is always dangerous to argue from analogy, but in many respects cirrhosis of the liver presents many analogies with cirrhosis of the kidneys. Thus the biliary form of cirrhosis (hypertrophic cirrhosis) of the liver would correspond with the ureteric cirrhosis of kidney obstruction, and the occurrence of malignant jaundice or acute yellow atrophy in the one case would correspond with the symptoms of acute uræmia in the other.

Portal cirrhosis of the liver would then correspond with arterial cirrhosis of the kidney. This analogy would suggest that cirrhosis of the kidney may be the result of some irritant in the blood, rather than of an anatomical change in the vessels. If this were the case, the question might fairly be asked, Why does the stress of the disease fall upon the kidney, and why do other organs escape? But we may ask the similar question in the case of cirrhosis of the liver. The analogy becomes still closer if it be remembered that there are widespread changes in chronic alcoholism—for example, in the heart, vessels, brain, nerves, &c., just as there are in granular kidney, and that cirrhotic liver is sometimes associated with cirrhotic kidneys; so that the same poison—for example, alcohol—does sometimes produce cirrhosis in both organs.

Yeld,* at St. Bartholomew's Hospital, in 131 cases of cirrhosis of the liver, found the kidneys granular in 44 = 33·5 per cent., Hawkins† gives 15 per cent., Kelynack‡ 18·5 per cent., Hamilton§ 21 per cent.

Moreover, the last stages of cirrhosis of the liver are not very dissimilar from those of the last stages of granular kidney, namely, well-marked cachexia, ending occasionally in acute toxæmia. As I shall show later, the resemblance between hepatic and renal toxæmia in both the acute and chronic forms is very close. Though each has its individual characters there is a striking family likeness between them.

Gout.

Gout may, of course, produce changes in the vessels. It is often associated with changes in the kidneys, and the association between gout and granular kidney is extremely close. The changes which long-standing gout is associated with in the vessels is usually of an atheromatous kind, and in elderly persons who have been the subject of gout for many years, interstitial nephritis is common, but the kidneys are not necessarily granular. In younger people, however, the conditions are different; there the changes in the vessels are not atheromatous, and the kidneys are

* 'St. Bartholomew's Hospital Reports' for 1898.
† Clifford Allbutt's 'System of Medicine,' iv, 173.
‡ 'British Medical Journal Epitome,' May 15th, 1897.
§ 'Pathological Society's Transactions,' ii, Part I, 283.

granular; so that there appears to be justification for the view which is held by some authorities that in young people with gout the kidneys are, with few exceptions, granular.

It is stated that in almost every case of granular kidney, whether the patient has had gout or not, urate of soda is found in some of the joints. This may be, however, but an expression of the obvious fact that the elimination of nitrogenous waste products is defective when the kidneys are diseased. The association between gout and granular kidney is at any rate very common.

But the coexistence of the two conditions does not establish the relation between them; for in the one case an elderly person who has long suffered from gout may as a consequence develop atheromatous changes in his vessels, which may lead to interstitial changes in the kidneys; while a younger person with granular kidney may develop gout in consequence of the renal disease. In other words, as I have already stated, gouty kidneys and granular kidneys are not necessarily the same.

Age Distribution.

Granular kidney is often said to be a disease of middle life, and this is true in the sense that the symptoms are usually evident then; in other words, the disease is then far advanced, and death may be near. The disease, however, must have commenced long before, perhaps years, so that even in these cases in middle life the onset of the disease is to be traced back, it may be, to youth.

Not only is the onset of the disease in many cases to be referred to early life, but the disease may even be advanced and fatal then. That granular kidney may occur in the young, even in children, is now generally recognised, but though rare it is still regarded as a much greater rarity than it really is. I have recorded an extreme instance fatal at the age of 18, and several instances in small children are recorded by Guthrie.

The following figures give the general age distribution above 20 years at the time of death:—

Table of Age at Death.

Age.	Dickinson.	Fagge.	Bartells.*	Galabin.	St. Bartholomew's Hospital (Woodbridge).†	Total.
About 20	1	—	4	2	2	9
20–30	17	—	4	6	10	37
30–40	38	18	9	12	19	96
40–50	73	39	4	19	43	178
50–60	55	36	7	21	41	160
60–70	43	24	5	16	28	116
Above 70	15	4	—	3	10	32

* One death at 18, one at 19.
† One death at 19, one at 20.

Diagram constructed from the combined statistics given, showing the Age Distribution at the time of death.

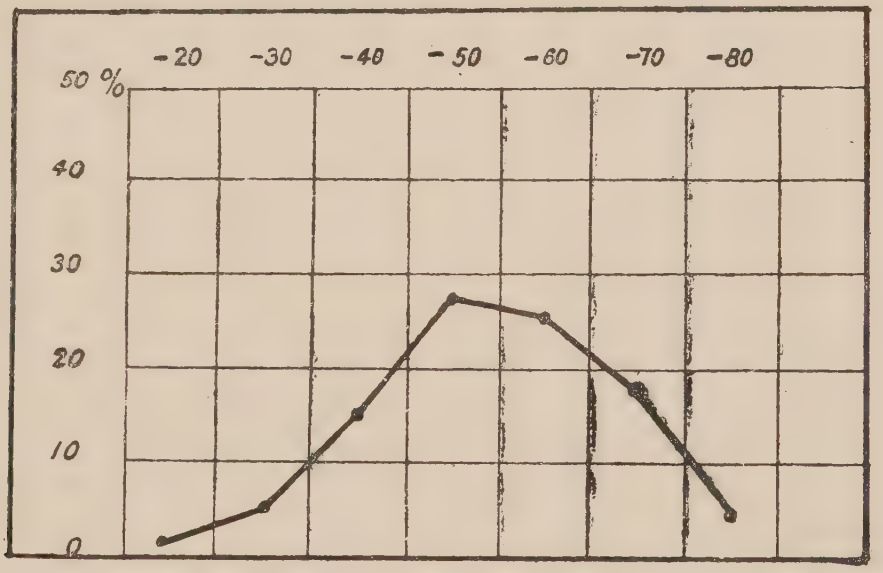


Table showing Age Distribution at the time of death of 206 cases at St. Bartholomew's Hospital.

20		20–30		30–40		40–50		50–60		60–70		70–80	
M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
3	—	6	9	11	14	33	26	42	14	27	9	7	5
3		15		25		59		56		36		12	

The Signs and Symptoms of Granular Kidney.

Granular kidney is a very insidious disease. For a long time it presents no symptoms at all, and can be recognised then by physical signs alone.

The only symptom perhaps in the early stage, and even this is not constant, is an increased frequency of micturition, especially at night; but as this has been of such gradual onset that the patient has become quite accustomed to it and does not regard it as peculiar, no complaint is made of it unless it be extreme, and the history of it is often only to be elicited on pointed questioning.

When definite symptoms arise the disease is already far advanced; in other words, the symptoms do not occur until late in the disease.

The symptoms fall, like the lesions, into two groups, namely, *cardio-vascular* and *renal;* and, speaking generally, the cardio-vascular are earlier than the renal.

The *cardiac* symptoms are those of heart failure, more or less pronounced.

The *vascular* symptoms are the more or less mechanical effects of the vascular lesion, and consist chiefly of hæmorrhage and its results.

The *renal* symptoms are the latest to develop, and fall into two groups according as they are of gradual development or sudden onset, and they are usually described as chronic and acute uræmia respectively. I should prefer to call them acute and chronic renal toxæmia. Both alike are to be connected with the wasting in the kidney and its consequent defective action.

Chronic Uræmia.—The gradual and progressive wasting of the kidney is attended with the gradual and progressive development of the chronic forms of uræmia. Renal cachexia is the name that I should prefer to give to chronic uræmia. It strongly resembles, both in character and course, the cachexia that is seen in a variety of other diseases, for example, in Addison's disease, in the later stages of cirrhosis hepatis, in diabetes, and malignant disease. It consists in a gradually increasing anæmia and asthenia, associated with various miscellaneous symptoms. The symptoms are similar in kind, differing only in the rate of development,

to those that are seen after complete obstruction of the ureters or removal of the kidneys, and have been experimentally produced in animals or observed in man as the result of pathological causes or surgical operation.

Acute Uræmia.—Similar as all the symptoms are in chronic uræmia they differ from those which are usually described as acute uræmia, and the difference is not one of degree only but of kind, so that they ought to be distinguished by different names; for the first I would reserve the term renal cachexia, for the second I should use that term which is generally accepted, namely, uræmia, and both alike, both the cachexia and the uræmia, may be of slow or sudden development.

I propose to deal with the signs and symptoms of the disease in this order :—

1. The physical signs.
2. The cardio-vascular symptoms.
3. The renal symptoms.

The Early Stages : the Physical Signs.

It is the early signs of the disease rather than the late symptoms that are of importance. What is required is an early diagnosis before the disease is far advanced, and this is essential if light is to be thrown upon the causes and course of the disease, or if influence is to be successfully exerted upon it by treatment.

In the early stage the diagnosis is to be made by physical signs and not by symptoms. The physical signs are high tension and thickened arteries, hypertrophy of the heart, and albuminuria. Where these are all present together the diagnosis is easy, even in the young, and the diagnosis thus made during life is verified *post-mortem*. But if one or other of these physical signs is absent, the question arises, Of what significance are those which remain ? For instance, if albuminuria be absent, what do high tension and thickened arteries mean in the young ? Or, if the arteries are not thick nor the tension high but albumen be present, what is the significance of this albuminuria ? Each of these questions is of great clinical importance and difficulty.

We know that the vascular changes, namely, thickened arteries and high pulse tension, develop so early in the course of granular

kidney that they seem to be—I do not say they are, but they almost seem to be—rather coincident, or, as some maintain, even antecedent, phenomena than effects consequent upon and produced by the kidney lesion. If this were so, vascular changes might be found without the kidney lesion, and such cases are described. I have seen what I believe to have been one case of this kind, but as the kidney has been lost, and I have not had an opportunity of re-examining it, I do not like to lay much stress upon it. The late Professor Kanthack, in discussing this subject with me shortly before his illness, told me he had a case of this kind in his collection, and would lend it to me for demonstration, but this, alas! failing me, I have no fresh facts of my own to bring forward.

Hypertrophy of the Heart.

Post-mortem evidence would point to hypertrophy of the heart as a most important sign of granular kidney, and so it is in the later stages, where the cardiac enlargement is such as to be easily made out. The heart may reach an enormous size. There is a case in the St. Bartholomew's Hospital Museum, in which the heart weighs 28 ounces.

In the early stages hypertrophy of the heart is of very little assistance in the diagnosis, because of the difficulty in recognising it then, especially if in slight degree only.

The frequency with which hypertrophy of the heart is found associated with granular kidney *post-mortem* is great, but at the same time it should be noted that it is not constant.

Goodhart gives 89·6 per cent., Ewald 84 per cent., Galabin 80 per cent., Kanthack and Holmes 90 per cent.

The value of these results is unfortunately depreciated by the almost constant omission of any reference to atheroma in the vessels, for atheroma will equally lead to hypertrophy of the heart. If atheromatous degeneration in the vessels ought to be distinguished, as I maintain, from the changes met with in granular kidney, hypertrophy of the heart ceases to be so important and conclusive a diagnostic sign as it has often been regarded.

The only signs upon which reliance can be placed for the diagnosis of hypertrophy of the heart during life are the increased area of cardiac dulness and displacement of the apex beat down-

wards and outwards; but in the early stages—that is, with hypertrophy of slight degree—these signs are not marked; and even in the late stages, when the hypertrophy is considerable, they are easily masked by the condition of the lungs. Considerable hypertrophy of the heart may be present and yet be incapable of demonstration by physical signs if, as often happens in granular kidney, it is associated with pulmonary emphysema.

The accentuation of the aortic second sound, which is sometimes described as if pathognomonic of hypertrophy of the heart, is really valueless, for it is met with wherever the arterial tension is raised from any cause whatever, and will be absent even in granular kidney when the tension falls. The same is true of the prolonged first sound, though not quite to the same extent. The hypertrophy of the heart is probably secondary to the vascular changes and the result of them, and therefore not of the same prime importance as they are, or so likely to be met with early.

Thickened Arteries.

Thickening of the arteries is one of the cardinal signs of the disease, and is never absent in advanced cases.

On this point some observations of Holst are interesting, in which he showed in 14 out of 15 cases of granular kidney the vessels of the skin were thickened, though not atheromatous.

Brailey's microscopic, as do Gunn's ophthalmoscopic, observations on the retinal arteries show the same thing.

Its Significance in the Young.

Of course the arteries grow thicker as age advances, as the result of atheromatous changes, and in elderly persons due allowance must be made for this.

But thickening of the arteries is by no means uncommon in young people, and the question as to what its significance is becomes important in relation to the early stages of granular kidney.

As the thickening of the artery must be estimated by the finger it is necessary to have some sort of guide as to what may be considered real thickening of the artery.

In a young person the healthy artery cannot be distinctly felt with the finger, and when pressed upon flattens out like a piece of

tape so as to be hardly felt; but if the artery be thick it can be rolled from side to side, and feels then like a piece of straw or rush, or even, when much thickened, like a pipe stem.

This thickening is real, and not apparent only as the result of increased tension, for the artery may be obliterated by one finger, and still felt by the other as thick as before, and when the tension is low, as it may be in the later stages, the thickening does not disappear.

There are, no doubt, physiological differences in the thickness of natural arteries, but, making allowance for this possibility, it is not uncommon to find the arteries so much thickened in young people as to be of necessity pathological, and it is thickening such as this that I say is by no means uncommon. Now, at this age the thickening cannot often be of an atheromatous nature, for although atheroma does occur in early life, it is very much rarer than is the thickening I am now speaking of. The change in these cases is no doubt muscular in great part, as it is in undoubted granular kidney, and the effect of nitrite of amyl upon the vessel is the same.

We are thus driven to this conclusion, that if it is not an early indication of granular kidney, then there must be some other form of arterial disease which we do not at present recognise. Arterial thickening in young people always rouses my suspicion, and leads me to look carefully for other evidence of granular kidney. Sometimes nothing more is found, and its significance continues doubtful, but that the change is pathological there can be no question. Still, the frequency with which in these cases the signs of granular kidney are either discovered on more careful investigation, or found to develop subsequently, leads me to the conclusion that the probabilities are in favour of this being really the early stage of granular kidney.

I have no actual figures to give in proof of its frequency, but the 8 per cent. which Mahomed gives as the frequency with which the high-tension pulse occurs would represent, to my mind, at least the frequency of thickened arteries, and it may be that we are both dealing with the same thing, for he describes the high-tension artery as rolling under the finger, but attributes this to high tension only. It is, however, really due to arterial thickening.

High Tension.

In well-marked (that is, advanced) granular kidney the high-tension pulse as well as the thickening of the arteries is well recognised. The sphygmographic tracing is characteristic; the curve has a round or square top, after which the line falls gradually. These peculiarities can be better felt with the finger; the slow, prolonged wave which the pulse makes under the finger is quite as evident as the fulness of the artery between the beats.

The contraction of the heart, which with healthy arteries causes a sudden rise—that is to say, produces a wave with a sharp and somewhat short crest—in granular kidney owing to the resistance which is offered to the circulation, instead of distending the artery greatly for a short time, spends itself in dilating the artery less for a longer time; thus the long wave, with its square or roundish top, is produced.

The tension is measured between the pulse waves; the arteries, therefore, feel abnormally full as well as abnormally tense between the beats.

When the heart begins to fail, the diminished force of beat does not necessarily alter the character of the wave. The artery may be less distended, and therefore the wave not so high, but its rounded top will still persist. The cardiac weakness may also, by diminishing the amount of blood driven into the arteries at each beat, reduce somewhat the tension, but in spite of that the pulse long retains its peculiar character.

When the tension falls, the fall is due to a failure of the artery, and not necessarily of the heart at all, though both may fail together. The low tension then observed is a neuro-paralytic phenomenon, and a bad sign; it occurs only in the later stages of the disease, but may then continue for some time. Though the tension may be low, the thickening of the arteries can, of course, still be easily made out, and is as characteristic as ever.

A persistently high tension, to whatever cause it may be due, is of itself mischievous, but not so with granular kidney, paradoxical as this may seem. The patient would be best without granular kidneys, but, having granular kidneys, is better with a high tension rather than low; in other words, the patient is worse when the tension is low; hence the use of digitalis, which does so much good in these cases by raising the arterial tension.

Low arterial tension, or rather the fall in tension, in the later stages of granular kidney is of great clinical importance, the value of which Sir William Broadbent has lately emphasised.

One other phenomenon I may refer to, which, though implied by what has just been said, is not generally recognised, although easy to be observed if looked for. I refer to irregular fluctuations in tension which take place in the later stages of the disease, before the tension becomes persistently low. The tension may vary considerably even in very short spaces of time—for instance, while the finger is feeling the pulse. These fluctuations are analogous to the flushings, the irregular respirations, and other symptoms of irregular vasomotor action which arise under similar conditions. They are very interesting, and may give the earliest indications of the neuro-paralytic failure which is approaching.

In the early stages of granular kidney, go back as far as we may, as soon as the disease is diagnosible the pulse tension is high and the artery thickened. The question then arises, what is the value of a high-pulse tension in a person in whom there is not yet other evidence enough to diagnose granular kidney by?

The clinical importance of high tension has been insisted on in valuable papers written by Sir William Broadbent* and Dr. Goodhart,† and if we go back 15 years we come to a series of papers written on this subject by the late Dr. Mahomed.‡ All alike directed attention to the clinical importance of the fact, though they do not altogether agree in their theories or conclusions.

Temporary or transient increase of tension may occur in a variety of affections, but it is not to these that I refer; I am dealing with those cases in which the tension is persistently raised. Now, Mahomed's view was that these were the early initial stages of granular kidney, and this I take to be also Goodhart's opinion.

I do not think Mahomed's work has been estimated at its true value. The term he introduced may have had something to do with this, for the expression, the pre-albuminuric stage, of which he considered high tension to be the chief indication, seemed to assume entirely what he had to prove. His work was based on careful clinical observation, and I believe that he was in the

* 'British Medical Journal,' August, 1883.

† Bradshaw Lecture, 1885, 'British Medical Journal,' ii, 327.

‡ 'Lancet,' January 11th, 1879, &c.

main correct, though possibly he attributed too much to high tension and too little to the thickening of the arteries to which I have referred. Even his own descriptions show that high tension was not the only factor in the pulse, but that the arteries were also thickened.

Sir William Broadbent, differing as he does from Mahomed in theory, yet attaches quite as great significance to the increase of pulse tension. He describes as the ultimate results of this increased tension a variety of symptoms—for example, cardiac failure, general disturbance of health, a sort of cachexia, and even hæmorrhage, which, as he stated, could be truly predicted long before it occurred.

These are the same symptoms as those produced by advanced granular kidney. If the kidneys are not granular in these cases—and of this no proof is given—the arteries must become diseased if hæmorrhage is to occur, for it can hardly be maintained that mere increase of tension can lead to the rupture of healthy vessels. If the vascular change is not atheromatous, nor associated with granular kidney, what is it?

So far as the results to which high tension leads, the views of writers on the subject are agreed; they differ only as to the relation of high tension to granular kidney. These differences in theory would remain a matter of opinion still, if there were not some fresh facts by which to elucidate them. These have been obtained by the more exact and careful study of the early eye changes met with in granular kidney.

As I shall show shortly, our knowledge of the early stages of albuminuric retinitis has been greatly extended of recent years. If in many of these doubtful cases of high pulse-tension and thickened artery albuminuric retinitis or the early changes which lead to it are found, and that even in cases in which albumen may not be present in the urine, the diagnosis of granular kidney will be surely justified. Considering that albuminuric retinitis is not invariably found even in advanced granular kidney, the fact that it can be discovered in these cases of doubtful nature is very significant and throws a light upon many of those other cases in which for the present the doubt must still remain more or less unsolved.

None of the writers referred to lay any stress upon eye-changes in their papers—at any rate, not as a means of diagnosis; but the

importance of these early retinal changes was not then so fully recognised as now. Some, at any rate, of these doubtful cases must be referred to the group of granular kidney, and the probability that a much larger number of them may be fairly so referred is thereby increased.

It appears to me, therefore, that there is presumptive evidence in favour of regarding these cases of high pulse-tension, as well as those of thickened arteries in the young, as the initial stages of granular kidney which we are in search of.

I do not wish to go beyond my facts or to speak dogmatically upon points which may still be open to question, but this appears to me the conclusion to which the facts so far point. At any rate, a larger number of these cases can now be proved to be granular kidney than has been hitherto supposed.

LECTURE II.

ALBUMINURIA.

Of albuminuria in granular kidney it is not necessary to say much. We know that it is usually small in amount, and that it is not constantly present. It may be found at special times only or may be absent for irregular periods, and might therefore be described as cyclical or intermittent respectively. It may be absent for long periods—even, it is said, altogether, though this is hard to prove. Its amount may be large—as much as half or more—but this generally depends upon some accidental complication, intercurrent nephritis or failure of the heart.

Given a clear case of granular kidney, these variations in the albuminuria do not cause surprise; but clinically the question often presents itself in a somewhat different form. In a given case albuminuria may be the chief symptom; we have then to decide whether granular kidney is the cause or not. It often happens that the evidence is not conclusive, for the patient appears to be in ordinary conditions of health. We are thus introduced to a problem of very great difficulty—that of so-called *physiological or functional albuminuria.*

For the present purpose we may regard the causes of albuminuria as falling into two groups, according as they lie in the kidney or outside it. In the latter case it may depend, on the one hand, upon some changes in the tissue or blood before the blood has reached the kidney, and may therefore be called prerenal; and, on the other, the urine may be normal when secreted, the albumen being added to it after secretion; these may be called postrenal. In each of these groups there may be some obvious cause or no cause obvious. Thus, the forms of albuminuria may be arranged into five groups:—

Albuminuria	Prerenal	1. Obvious cause — for example, morbus cordis, fevers, &c.	Physiological.
		2. No obvious cause	
	Renal	3. No evident renal disease	
		4. Evident renal disease.	
	Postrenal	5. From urinary passages, generative organs, vagina, &c.	Accidental.

Groups 2 and 3 are embraced by the term physiological albuminuria.

This table draws attention at once to the difficulties which have to be dealt with before the diagnosis of physiological albuminuria can be safely made.

The postrenal group is of great importance, for care must always be taken to exclude all accidental contamination, especially such as would result from the presence of cystitis, gonorrhœa, or from the admixture of discharges from the uterus and vagina, or from the prostate and vesiculæ seminales.

Experimentally, albuminuria has been produced in perfectly healthy persons by excessively albuminous diet, by violent muscular exercise, by cold bathing and exposure to cold; but even then it is probably not capable of being produced in every person, or at any rate not with equal ease, so that even in such cases some other factor is necessary.

Again, in an apparently healthy person albumen may be found occasionally present under certain circumstances, as, for instance, on rising in the morning or after food; and the albuminuria has then been called postural, dietetic, cyclical, or intermittent respectively. Intermissions or variations in the occurrence of albuminuria are no evidence that there is no disease of the kidneys, for they are frequently observed in persons recovering from acute nephritis in whom it is by no means uncommon to find albumen absent, it

may be, for the greater part of the day, and present only at certain times; in other words, the kidneys may be affected, and yet albuminuria be only intermittent or cyclical. This is, of course, well known to be the case in granular kidney. Even acute nephritis in slight degree may be very easily overlooked, especially in children, and when œdema is absent the nephritis may not even be slight, as the presence of blood and epithelial casts shows.

Admitting all these exceptions, there remains a group of cases in which albuminuria is present, with apparently nothing whatever in the previous history or the actual condition of the patient to explain it. The choice of a term by which these cases can best be described is difficult. Latent albuminuria is, perhaps, that to which least objection can be raised, but it is often not so much latent as overlooked. Early albuminuria is open to the two objections, either that it might be thought to refer to albuminuria only in the young, which would be incorrect, or that it implied the opinion that it was an early stage of disease, and this again would be begging the question. Albuminuria in the apparently healthy, on the whole, seems to me to have least objection to its use, and to state the facts best.

Albuminuria in the Apparently Healthy.

The statements made by different observers as to the frequency with which albuminuria occurs in the apparently healthy vary greatly.

Thus, Leube's * observations upon presumably healthy young adults, which are so often quoted, yield 30 per cent. Fürbinger's † observations upon children yield 10 per cent. Goodhart's ‡ observations for all ages on the contrary only 2·6 per cent., while more recent observations upon boys yield more than 22 per cent. Finally, Possner states that by sufficiently delicate tests albumen may be shown to be present in the urine of everyone.

Variations so considerable as these in observations made by equally competent observers show that there must be a fallacy

* 'Virchow's Archiv.,' 1878.
† 'Zeit. f. klin. Med.,' xiii, 1, 1887.
‡ 'British Medical Journal,' May 17th, 1890.

somewhere, and it may lie in the fact that different sets of cases have been observed or different tests employed.

To consider Possner's statement first. There is no doubt a certain shedding of epithelium in the genito-urinary passages of everyone; if, then, a large quantity of urine be taken, and, after concentration, treated in elaborate ways, it is evident that the albumen will be detected which is derived from the normal epithelium, and which must be present to a greater or less extent in the urine of everyone. Whether this be the origin of the albumen or not, so far as Possner's observations go, they amount to no more than this, that by special methods a minute amount of albumen may be detected in everyone's urine. This, however, is not what is meant by the clinical term albuminuria.

Looking broadly at the figures given by the different authors, these facts are evident:—

1. Most of the observations upon which the statements are based have been made upon children and adolescents.
2. And those almost entirely of the male sex.
3. That the higher percentages have been obtained by including cases in which very minute traces of albumen were found by the most delicate tests.

In respect of *sex*, the choice of males has probably been chiefly determined by convenience; at any rate I do not think sex has any influence upon the frequency of albuminuria, although the question has not, I believe, been worked out specifically from this point of view.

In respect of *age*, Heubner* states that of the cases published, 77 per cent. were in persons under 20 years of age, and 39 per cent. in children, and to this I may add that hardly more than 10 per cent. at the most were in persons above 25.

Roughly speaking, the cases thus group themselves into two periods:—

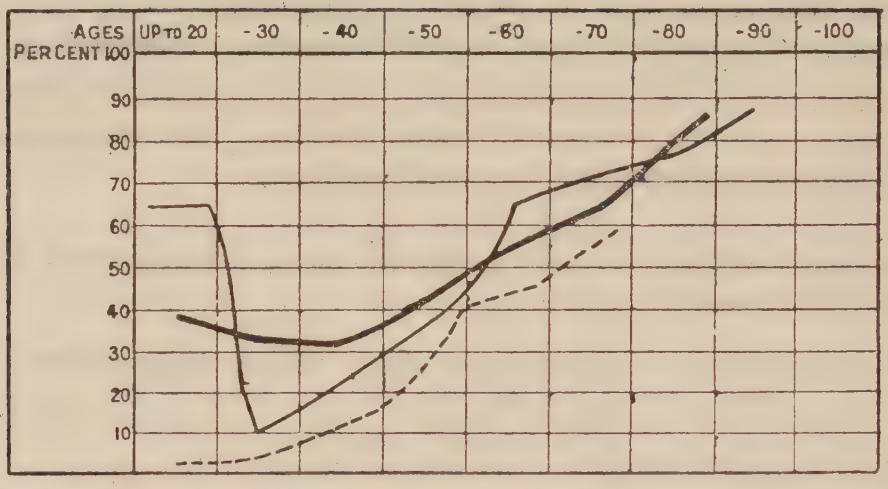
1. Those before the age of 25, and
2. Those after 25.

The difference between these two periods is indicated upon the diagram, which shows the remarkable frequency of albuminuria before the age of 25 years, the drop which occurs about that age, and the gradual rise with each age-period after that age. The diagram also indicates that the conclusions which apply to the

* Virchow's 'Jahrb.,' 1890, ii, 722.

one age-period cannot, without due allowance, be applied to the other.

Diagram Constructed from the Collated Statistics given by Stirling (Light Line) and Levison (Thick Line).



The dotted line shows the frequency of granular kidney in proportion to healthy kidneys, derived from Mahomed's figures.

Forms of Albumen and Tests.

There are many forms of albumen, but we need only consider two forms :—

1. Serum-albumen, which for our purpose will also include serum-globulin; and
2. Nucleo-albumen, a mucin-like body, which was formerly confused with mucin.

It is by reckoning nucleo-albumen in with serum-albumen that the high percentages of albuminuria have been obtained.

Serum-albumen and nucleo-albumen both alike are precipitated by heat and acetic acid, by salicyl-sulphonic acid, and by picric acid.

The distinguishing tests are these :—

1. Nucleo-albumen gives with acetic acid and dilution a distinct cloud, while serum-albumen gives at most a haze or nothing at all.

Acetic acid alone precipitates both albumen and nucleo-albumen, the former less rapidly ; but on dilution the nucleo-albumen reaction is not diminished, or may be somewhat increased, while the haze with albumen becomes less distinct in proportion to the dilution.

2. Heller's test gives a distinct line with albumen at the junction of the

two fluids. But with nucleo-albumen the ring forms ½ to 1 centimetre above the junction, or, if there is no ring, a haze is produced throughout the solution.

3. A saturated solution of sodium chloride with acetic acid, when heated, gives a faint reaction with nucleo-albumen but a distinct one with serum-albumen.

4. Ferrocyanide of potassium and acetic acid, which is very sensitive to serum-albumen, gives a reaction with nucleo-albumen only if it be present in considerably more than a trace.

5. A saturated syrupy solution of citric acid precipitates nucleo-albumen much more readily than serum-albumen. The ring method, as in Heller's test, is the most satisfactory way of making this test.

Mucus also gives some of the above reactions—for example, with heat and acetic acid, salicyl-sulphonic acid, and picric acid, as well as acetic acid in the cold, but it is present only in very slight amount. It does not give the ferrocyanide of potassium test if performed in the right way—that is to say, if the urine be added to the ferrocyanide and acetic acid solution, and not, as the test is often made, by adding the ferrocyanide solution to the urine or by adding acetic acid first.

There is no single test upon which reliance can be placed to distinguish absolutely between nucleo-albumen and serum-albumen, but these tests systematically used in order will suffice to make the distinction quite clearly in most cases.

Desiring to form my own opinion upon this question, I asked Mr. Levison to undertake some observations for me. He has done the work with very great care, and his paper will shortly be published *in extenso;* for the present I shall only give the more important conclusions.

He examined 336 healthy persons, of whom 108 were young soldiers; the rest were hospital convalescents.

The cases were thus grouped:—

		Below 25.	Above 25.
Males	254	155	99
Females.. ..	82	31	51
Total ..	336	186	150

A proteid reaction of some kind was obtained in 136, or 40·47 per cent., serum-albumen occurring in 22·02 per cent., and nucleo-albumen in 18·05 per cent.

The figures are set forth in the following table :—

——	Below 25.	Above 25.
Proteid reaction of some kind in 136 cases =40·47 per cent.	5 cases	61 cases
Serum-albumen in 74 cases=22·02 per cent...	42 „	32 „
Nucleo-albumen in 62 cases=18·44 per cent...	33 „	29 „
Negative in 200 cases	112 „	88 „

Making allowance for the different numbers in the two groups—that is, above 25 and below 25, the percentage was almost exactly the same in each.

The amount of albumen in all cases was extremely small, and probably in quite half the cases would have been entirely overlooked in the ordinary methods of examination. The largest amount in these series was not more than would give a ring of 1 to 2 millimetres in thickness with nitric acid.

Nucleo-albumen was found alone in nearly one-half of the cases—18·44 per cent., and this was the same in both the groups—that is, before 25 as well as after 25. It is probable, therefore, that in Leube's results, as in these, one-half were due to nucleo-albumen.

Of the cases in which serum-albumen was present (22 per cent.), in 12 per cent. the amount was quite minute, and only in the remaining 10 per cent. would have been easily recognisable by the ordinary methods.

No clinical difference could be detected between the cases with serum-albumen and those with nucleo-albumen, nor should any be expected if it be true that nucleo-albumen is but a salt of ordinary albumen with chondroitic-sulphuric acid or nucleic acid, which are constituents of normal urine.

The age distribution of the cases examined is shown in the following table :—

Age-Period.	Total Number of Cases Examined.	Albumen : Serum-A. Nucleo-A.	Percentage to Cases at each Age-Period.
1–5 years	2	0	0·00
5–12 ,, ...	18	2	11·10
12–18 ,, ...	37	14	37·80
18–25 ,, ...	129	59	45·70
25–35 ,, ...	47	17	36·10
35–45 ,, ...	32	11	34·40
45–55 ,, ...	37	15	40·50
55–65 ,, ...	22	11	50·00
65–75 ,, ...	11	6	54·10
Above 75 years	1	1	100·00
Total	336	136	40·37

These observations show that the great frequency of albuminuria in apparently healthy persons is not overstated if all those cases be included in which there is the minutest possible trace evident only on careful examination, but inasmuch as in most of these cases, probably one-half, the trace is so small that it can only be detected with great care when specially looked for, the contrast between these results and the general impression obtained from ordinary clinical experience is not so great as at first sight it appears to be.

It is evident that the conclusions drawn as to the frequency of albuminuria in one age-period must not be applied without due allowance to any other age-period. Speaking generally, the significance of albuminuria is entirely different before 25 and after 25.

What is required is a systematic series of observations in which the same carefully-selected tests have been employed, and a sufficient number of cases of the one sex and of the other examined at the different age-periods :—

1. In infants up to the age of 5.
2. In young children from 5 to 10.
3. In boys or girls from 10 to 18, that is, the school age
4. In young adolescents, 18 to 25.
5. In young adults, 25 to 30.
6. In the middle period of life, that is, from 30 to 50.
7. In the later periods of life, over 50.

1. *Infancy and Early Childhood.*—Of albuminuria in infancy and early childhood we have not much information. In new-born infants albuminuria is stated to be common, but that need not be a matter of surprise. In babies up to a few months old, Holt quotes Cruse as having found albuminuria in 28 out of 90 cases (31·1 per cent.).

2. *From Infancy up to the Age of* 10.—Albuminuria is said to be less frequent up to the age of 10. Ward * examined 120 cases between the ages of 5 and 12 years, but his results bear this statement out fairly well, for he obtained a reaction with heat and acetic acid in 29 (24·16 per cent.), but the amount was always minute and sometimes the merest trace. In only nine cases (that is, 7·5 per cent.) was the amount appreciable.

3. *From* 10 *to* 18 *years—that is, Children during School Age.*—At this period statistics abound and show the extraordinary frequency of albuminuria.

The following are some of the figures:—

Stirling	369 boys	....	20·8 per cent.	
Ribbert	240 „	....	22·0 „	
		summer (104 cases)	29·8 „	
		winter (136 cases)	16·0 „	
Dukes	—	many years' experience	22·0 „	at least
Grainger Stewart	100 boys	....	17·0 „	

Stirling's table shows a gradual increase in frequency from the age of 12 to 15 or 16.

——	Age.				
	12 years.	13 years.	14 years.	15 years.	16 years.
Number of cases ..	20	59	88	147	55
Percentage.. ..	5·0	10·2	16·0	29·4	23·6

Although the amount of albumen is usually small it may be considerable.

* These observations were made in Dr. Garrod's out-patient department in the Hospital for Sick Children, Great Ormond Street.

Thus in a specimen which was kindly sent me recently for examination by Dr. Clement Dukes the amount of albumen was as much as 2 per cent. —that is, 20 grammes in the litre. This was in the morning urine. In the evening, after a day's rest in bed, the albumen was not more than a trace, and this chiefly nucleo-albumen. In a third sample, after the boy had got up again, the amount of albumen was nearly as large as before —namely, 1·4 per cent., and about $\frac{1}{8}$ = 0·18, was serum-globulin and nucleo-albumen. This specimen was centrifugalised, and showed a few granular and epithelial casts and some epithelial cells.

It has been suggested that the presence of albuminuria at these ages is due to the active development of the sexual organs, and probably also to the presence in the urine of discharges derived from these organs, and that the form of albumen is generally nucleo-albumen. The albumen is, however, generally serum-albumen and not nucleo-albumen, and there is no proof that nucleo-albumen is derived from the urinary passages and not from the kidney. Nor is there any proof that masturbation is a predisposing cause; indeed, what evidence there is, is against it.

4. 18 *to* 25 *years, Adolescents.*—It is upon cases of this period that most of the statements as to physiological albuminuria in adults are based.

Most of the observations have been made upon young soldiers :—

Leube	119 young soldiers	....	20·4 per cent.	
Grainger Stewart	205	„	„	.... 27·6 „
Tieman	186	„	„	.... 27·4 „
Zeehuisen	144	„	„	.... 14·6 „
Levison	108	„	„	.... 47·25 „

The 108 young soldiers that Levison examined ranged from 17 to 27 years of age, but of these only eight were above 25.

A proteid reaction of some kind was found in 51 cases = 47·25 per cent.

This was due to serum-albumen in 30 cases = 27·75 per cent.
This was due to nucleo-albumen in 21 cases = 19·50 per cent.

In all the nucleo-albumen group and half the serum-albumen group the quantity present was very minute = 33·4 per cent., that is, in only 14 per cent. was the quantity certain to have been detected by ordinary testing.

In three of the latter cases in which a moderate amount of albumen was found, careful microscopical examination was made after centrifugalising, and nothing discovered except a few epithelial cells.

The effect of exercise is well shown thus :—

In 29 Cases after Drill.	In 79 Cases without Drill.	24 New Recruits who had not been Drilled.
Proteid of some kind in 18 cases = 62·3 per cent.	In 33 cases = 41·7 per cent.	5 cases = 20·75
Serum-albumen in 10 cases = 34·48 per cent.	In 20 cases = 25·31 per cent.	3 cases = 12·54
Nucleo-albumen in 8 cases = 27·82 per cent.	In 13 cases = 16·45 per cent.	2 cases = 8·25

Thus morning drill considerably increased the total frequency of albuminuria, but made no difference in the relative frequency of the two kinds of albumen.

5. 25 *to* 30 *Years.*—For this period there are very few published statistics. By the kindness of friends I have been enabled to obtain the experience of some life offices relative to the occurrence of albuminuria in applicants for insurance presumably healthy below the age of 30 years.

At the Provident Clerks' Life Assurance Association I find that in the course of the five years 1893 to 1898, 7,950 policies came before the office, and 4,488, that is to say, a good deal more than half, were persons under 30 years of age; out of this number, 25 cases were found to have albumen in the urine. This was a percentage well under 1, actually of 0·55; of this number no fewer than 18, that is, two-thirds, were under the age of 25.

At another office, for the period of 10 years, the number of applicants below 30 years of age was, roughly speaking, about 750, and out of these seven only were deferred or rejected on the ground of albuminuria; this gives a percentage of nearly 1.

No doubt more precise examination would have raised this percentage somewhat; still, I think the facts are sufficient to show that at this age-period albuminuria is much less common than in any precedent period, and that it probably stands at its lowest point in life.

6. *Above* 30 *Years.*—There are a great many figures to show the general frequency of albuminuria for all ages during the insurance period, but they tell us little of what we really want to know—

namely, the relative prevalence of albuminuria at the different age-periods throughout adult life.

Thus Washburn,	for	1,000 cases,	found	3·5 per cent.
„ Munn	„	1,000	„	11·0 „
„ Tieman	„	2,000	„	9·0 „
„ Anonymous	„	6,400	„	7·1 „

To these we may add as derived from general sources :—

Grainger Stewart	100 adults	10·0 per cent.
Goodhart	1,600 out-patients	2·6 „

With these we may compare Levison's figures, which yield a much higher average percentage, equal to 40 per cent.

Petersen's results give the key to the difference in the percentages obtained.

He examined 1,000 soldiers and recruits by Heller's test only, and found albumen present in the morning urine, on rising, in 3·79 per cent. ; at 11 a.m., after three hours' drill, 15·11 per cent. ; at 6 p.m., after two hours' drill, 9·02 per cent.

The difference in results is evidently due to the different tests employed.

It would greatly simplify matters if we could agree upon a standard test, and the best, in my opinion, would be Heller's, *i.e.*, the cold-nitric-acid-ring-test. It is sufficiently delicate, for it will show one part in 10,000 at once, and one part in 35,000 in four minutes. An amount of albumen which cannot be shown by Heller's test may, *quâ* albumen, be disregarded clinically.

The Significance of Albuminuria in the Apparently Healthy.

Albuminuria in the apparently healthy, apart from its scientific interest, has a practical bearing—in boyhood as affecting the school career, in young men as affecting the chance of appointments, and in adult life as affecting insurance.

In considering the significance of albuminuria, it will be well to retain the division into two groups, before and after 25, as we have hitherto done.

Significance in Boys and Adolescents.

Albuminuria is common during the period of growth and immaturity, and becomes less frequent when development is approaching completion.

The question arises whether we can connect these two facts together. Assuming that during the period of growth all the tissues of the body are in a condition of instability and immaturity, it is easy to suppose that very slight causes might disturb their balance, and thus in the case of the kidneys albumen might appear in the urine as the result of causes which would have no effect upon the fully-developed or adult organism or organ.

What the clinical significance of this albuminuria in childhood and adolescence is can only be determined by clinical observation, by carefully investigating the cases at the time, and watching them over a long period.

The boys in whom albuminuria is found appear to fall into at least two groups. In one the lad appears to be robust and healthy, has a rigid artery and a high tension. These are the cases which Clement Dukes has recorded as met with so frequently in schoolboys. In the other the lad is not robust and healthy, but feeble and pale; the tension is low and the artery not thickened. These two groups probably indicate not only different conditions but a different prognosis.

In no case is the albuminuria strictly speaking physiological; it is always pathological, though not necessarily renal. What we require to know is the course which these cases run in after-life.

If albuminuria is so common in youth and adolescence, and becomes so much less frequent in early adult life, it follows that many of the cases have no very serious significance—in other words, that they get well. This, indeed, we also know from direct observation. Yet with those who have most experience there is evidently still some lurking doubt about the ultimate result in not a few instances.

Granting that many, perhaps the majority, ultimately recover, there remains another group in which the patients continue to pass albumen for many years and yet remain in good health, and a third in which signs of renal disease ultimately develop.

Johnson* described a case in which recovery took place after seven years of persistent albuminuria, and other cases are recorded in which the albuminuria persisted for 10, 15, or even 20 years, without other evidence of disease appearing. Saundby† gives the case of a man rejected for insurance on account of albuminuria who was in good health 18 years afterwards.

Yet, as will be shown later, maintenance of health for many years is not incompatible with damaged kidneys and premature death.

Making all allowance for exceptional cases such as these, the significance of albuminuria in early life must for the present still remain doubtful.

Clement Dukes says in one of his last writings on the subject:—"Familiarity with the disease has not bred the proverbial contempt, for I am convinced that a number of these cases never recover. In many, indeed the majority, the albuminuria is transient and never recurs; in others it becomes persistent from a frequent repetition of transient attacks, from neglect or imprudence, or from failure of accurate diagnosis, and eventually results in chronic disease of the kidney."

All these cases must be carefully watched for a long time before nephritis can be positively excluded; the more constant and persistent the albuminuria is the greater the probability of renal disease, and the longer it continues the more serious the outlook.‡

Significance in Adult Life.

This is important in relation to life insurance.

Before 25 many cases may be curable; after 25 the chances of renal disease increase rapidly. Conclusions drawn from cases under 25 cannot be applied without consideration to cases over 25.

The diagram already given (p. 138) shows, first, the frequency of albuminuria at the different age-periods after 25, and, secondly, the relative frequency of granular kidney after 25.

There is a great difference of opinion professionally as to the significance and importance of the traces of albuminuria met with in otherwise apparently healthy persons applying for insurance, and I think there is also much difference in practice.

* 'British Medical Journal,' ii, 1879.

† *Ibid.*, May 10th, 1879.

‡ *Cf.* Holt, 'Diseases of Children.'

It would be important, therefore, to trace these cases in order to ascertain what became of them in after-life. In some instances this has been done.

Thus, in Washburn's series, out of 1,072 cases, 38 had albuminuria—that is to say, 3·55 per cent. Of these 38 cases, 18 were traced; 14 more of these cases were known to be living, but the condition of the urine was unknown, and six were lost sight of. Of the 18 cases traced, six were stated to have recovered; six died of renal disease; five others had albumen still some years after; and one died of phthisis. Thus, out of 18 cases, six had renal disease for certain and five more probably, giving a percentage of not less than 33, and possibly of 60. Washburn calculated that the mortality rate among these cases for the age was 17·54 per cent.—that is to say, was nearly double the normal average.

Munn found that, out of 25 cases, one died within the year following the examination, and four more died during the subsequent three years, while in all the rest the health had obviously deteriorated.

Rabagliati's results bear, perhaps, only indirectly upon the question we are discussing. He showed in connection with the Scottish Widows' Fund that, of 3,246 deaths, 255 were referred to urinary diseases—that is, 7·8 per cent., and of these, 4·3 per cent. were due to diseases of the kidneys and nephritis. During the same period in Bradford the general mortality from urinary diseases of males above 15 yielded the rate of 3·4 per cent. Thus, the insured lives had a mortality rate for kidney disease much above the normal. He further states that the average age of albuminuria at death was 57.

We may thus conclude that, in spite of all ordinary care and consideration, a considerable number of renal cases slip through the medical examination; and hence the importance of not minimising the evidence, even when it is slight, if any early renal mischief is apparent.

From another office I have a very valuable report, to the author of which I should like to acknowledge my indebtedness, but I am prevented by the condition upon which the information is furnished me, so that, while I can vouch for the figures, the report must remain anonymous.

In a series of 6,400 adults examined between the years 1877 and 1893, 454 cases of albuminuria were found. Of 137 cases among these in which the urine was examined microscopically, casts were found in 31—that is, in about 23 per cent. Thus, in round numbers, albumen occurred in about 7 per cent. of all apparently healthy adults; the probability of finding casts in the urine associated with albumen was about 1 in 4.

Some of these cases have been traced in after-life, and the following figures have been furnished me :—

Albuminuria in Adults Apparently Healthy.

Very few were examined microscopically. The specific gravity ranged from 1,034 down. Personal and family history and build were good as a rule, but some cases have been admitted in which one or more of these factors have been under the average.

TABLE I.

	A. Ages between 20 and 39.		B. Ages 40 and over.	
	Living at end of	Died during	Living at end of	Died during
1st year	194	2	120	4
2nd ,, ...	193	1	115	5
3rd ,, ...	188	5	113	2
4th ,, ...	187	1	109	4
5th ,, ...	181	5	106	3
6th ,, ...	174	7	101	5
7th ,, ...	166	5	89	12
8th ,, ...	147	6	78	2
9th ,, ...	99	2	53	3
10th ,, ...	44	—	27	2
11th ,, ...	22	1	13	2
12th ,, ...	16	1	10	—
13th ,, ...	11	—	5	—
14th ,, ...	8	—	3	—
15th ,, ...	5	—	1	—
16th ,, ...	2	—	—	—

Among these cases 49 were stated to have had casts, and the following table gives the results in these :—

TABLE II.

	A. Ages between 20 and 39.		B. Ages 40 and over.	
	Living at end of	Died during	Living at end of	Died during
1st year	25	—	22	2
2nd ,, ...	25	—	20	2
3rd ,, ...	24	1	18	2
4th ,, ...	24	—	18	—
5th ,, ...	22	2	18	—
6th ,, ...	21	1	16	2
7th ,, ...	20	1	13	3
8th ,, ...	20	—	12	1
9th ,, ...	17	1	10	1
10th ,, ...	12	—	5	1
11th ,, ...	5	1	4	—
12th ,, ...	2	1	3	—
13th ,, ...	2	—	3	—
14th ,, ...	2	—	—	—
15th ,, ...	1	—	—	—
16th ,, ...	1	—	—	—
17th ,, ...	1	—	—	—

Table I.—For the first eight years or so nearly all the cases were traced, and the record is remarkably complete; after this many apparently were lost sight of. During those eight years the total mortality in Group A, that is, in cases under 40, was 17 per cent., and in Group B 30 per cent., a very considerable death-rate for presumably healthy lives. Evidently, therefore, the albuminuria largely increased the risk.

But the table also shows that some of the cases lived a long time. Thus, in Group A, at least 44 persons, that is, 22 per cent., were known to have lived 10 years, and probably several others were alive who had been lost sight of. Sixteen were known to be alive after 12 years, eight after 14, and two at least lived more than 16 years. Similar facts are shown in Group B.

Table II.—In this class, taking eight years also, the mortality was still higher—namely, 20 per cent. in Group A, and 50 per cent. in Group B. Still, here again some individuals were known to have survived for many years.

The following table has been calculated for me by an actuary friend, to show approximately, as far as the figures permit, the difference in mortality compared with healthy males, and to make the results more obvious they have been taken for differences of two years in age :—

Ages Attained.*	Rate of following Year.		Ages Attained.	Rate of following Year.	
	Rate from Figures Given.	Rate for Healthy Males.		Rate from Figures Given.	Rate for Healthy Males.
30	—	—	50½	3·70	1·97
32	1·56	1·06	52½	2·64	2·20
34	3·27	1·11	54½	3·74	2·43
36	3·33	1·16	56½	7·13	2·69

* Starting with average of those given, and assuming uniform increase for eight years.

Without laying any stress upon the actual figures these calculations show how largely the presence of albuminuria raises the mortality, as well in those below 40 as in those above.

We must conclude, therefore, that for the ages at which life insurances are generally effected, that is, from 25 years upwards, the presence of even a trace of albumen in the urine is of considerable significance. It is more and more likely to mean disease as age advances, and therefore the risk to the office continually increases. Even at the earlier periods—from 25 to 35—it is a question whether these lives should be accepted even with a considerable addition, owing to the difficulty of excluding the commencement of granular kidney.

Rabagliati threw his conclusions into the following form :—(1) over 40, reject; (2) under 40, load heavily; (3) in young adults, rate up moderately.

With the first recommendation I entirely concur. The second is necessary, but the loading might be prohibitive and tantamount to rejection for any age over 30. In respect of the third, between 25 and 30 there would have to be a fairly heavy loading, but

between 18 and 25 many cases could be safely accepted without any great risk.

Each case of this kind, however, must be considered on its merits. Albuminuria is not the only fact to be considered, suspicious as it may be. At each age there may be innocent and harmful albuminuria, and it is the business of the physician to distinguish, if possible, between them.

I do not know any class of case which presents greater difficulties to the medical adviser to the life office, or affords greater scope for good judgment and skill; so that while rightly appraising the risk to the office on the one hand, he should at the same time advise what is fair and just to the proposer.

Conclusions.

The general conclusions to which these considerations lead are these:—

1. That the so-called physiological albuminuria is always pathological, even if not always renal, when the amount of albumen is more than the merest trace.

2. It is probably pathological, even in those cases in which the amount of albumen is but a trace, and no cause obvious.

In this respect it is interesting to recall the experience of Eales, who, out of 14 cases sent him as instances of physiological albuminuria, found no fewer than five with marked albuminuric retinitis, showing that what he had to do with was not even physiological albuminuria at all, but well advanced kidney disease.

It is clear, therefore, that in every case the patient must be carefully examined, every possible cause of albuminuria searched for, and every other condition in the patient noted.

If nothing else be found and the albuminuria exist alone, it may possibly be harmless or accidental, and of no serious significance.

If renal derivatives are found as well as albumen the chances of renal disease are greatly increased.

Even if no renal derivatives are found, the kidneys are in all probability granular if the arteries are thickened and the pulse tension high.

ALBUMINURIC RETINITIS.

Albuminuric retinitis brings me to another group of signs, viz., those of the later stage; for, typical as albuminuric retinitis is, it is of little help in the early diagnosis of granular kidney, as it only occurs late in this disease, when the arteries are degenerate and the kidney mischief far advanced.

In its typical form albuminuric retinitis is characteristic and pathognomonic, *i.e.*, occurs in no other condition except that of chronic renal disease. In some stages, especially in the exudative or inflammatory form, it may closely resemble the optic neuritis met with in intracranial disease, *e.g.*, cerebral tumour, but in such cases its importance is not so great, for the diagnosis is generally clear on other grounds.

Yet upon many important points the statements of different authorities are very conflicting and difficult to reconcile. This, I think, could be explained if it were true that there were different forms of albuminuric retinitis with different pathological histories. No attempt, however, is usually made to distinguish between them or to connect them with the different forms of renal disease.

Kidney diseases fall into two great groups, according as dropsy is a prominent symptom or not. Granular kidney may be taken as a type of the one group and the various forms of tubal nephritis as the type of the other. The kind of albuminuric retinitis most frequently met with in these two groups will, I think, be found to be different. Thus, the exudative and inflammatory form will be most frequently met with in those cases in which dropsy predominates, *i.e.*, in parenchymatous nephritis, and the degenerative and hæmorrhagic form in that in which dropsy is absent or accidental, *i.e.*, in granular kidney.

The lesions of albuminuric retinitis consist of white patches, hæmorrhages, and exudation variously combined with each other, and, lastly, quasi-inflammatory conditions.

It would seem from some of the statements on the subject as if these were but different stages of an affection culminating in the last, or, as it is often called, the most extreme form. Yet this is by no means the case; for the last form is often independent of the others, is frequently of very rapid onset and acute development, and, extreme as it is, may disappear without a trace.

The Exudative (Inflammatory—Extreme) Form.

In this form the changes are wide-spread and extreme. Thus, the disc may be greatly swollen, the edge indistinct and frayed out, and the whole retina hazy, as the result, no doubt, of œdema. White patches, more or less bright, of varying size and irregular shape, are scattered over the whole central part of the retina, especially round the disc and yellow spot. They are due to exudation in the deeper parts of the retina. The vessels are often visible as they run over the white patches, and are generally not so much covered with effusion as is the case in other forms of neuro-retinitis. The veins are greatly distended and the arteries small, while hæmorrhages are common, both flame-shaped and irregular. In fact, this form closely resembles what is seen in other forms of acute neuro-retinitis, and may in some cases introduce difficulties of diagnosis.

Thus I have recorded two cases in which the resemblance to cerebral tumour was so close that the diagnosis was not certain until the patient had been under close observation, the symptoms in both cases being violent headache, vomiting, giddiness, loss of sight, and well marked neuro-retinitis.

Yet in most cases there is something in the condition of the eye to suggest the true nature of the disease.

Speaking generally, so far as I can judge at present, this form of albuminuric retinitis is rare except in the parenchymatous group of renal disease, *i.e.*, in the acute and chronic tubal forms. I think it may be connected with renal dropsy, and as renal dropsy is not very common in granular kidney, so this form of albuminuric retinitis but rarely occurs in this disease, but is more frequently met with in the other group, viz., that of parenchymatous nephritis.

We do not know why it occurs in these cases, for it is not simply associated with dropsy, *i.e.*, the direct result of the dropsy, for, speaking generally, it is rare even in parenchymatous nephritis when the dropsy is extreme, and it may be met with, too, when the dropsy is only moderate. Again, it is rare in acute cases, or in the early stages, of parenchymatous nephritis, though it is in these cases that the dropsy is usually most marked, while it is not so rare in the later stages, *i.e.*, in the chronic cases. In this form it is, I believe, always bilateral; and taking this

fact into consideration I think the most probable explanation of its origin is that it is a subacute toxic inflammation.

One thing is remarkable considering the amount and extent of the exudation, viz., that detachment of the retina in these cases is comparatively rare. Most of the instances recorded, and the only ones I have myself seen, have occurred not in the acute cases at all, but in granular kidney, where there was but little or no general dropsy.

In the cases of chronic parenchymatous nephritis, there is often fibrosis, in most patchy and not extensive, but in some widespread, or even general throughout the kidney, and leading to considerable thickening of the capsules of the malpighian bodies. Yet I do not think we should be correct in associating the retinitis in these cases with the fibrosis.

Although if albuminuric retinitis occur at all in chronic parenchymatous nephritis, it is, I believe, generally of the more extreme exudative form, still it may be restricted to a few small white patches near to the yellow spot closely resembling those of granular kidney, yet generally rather woolly grey in appearance than pearly white. These may be regarded as the early stage of this form of albuminuric retinitis.

Complicated as the question is, we seem to be justified in drawing these conclusions: that this form of albuminuric retinitis is of an exudative, inflammatory, type; and that it is probably of toxic origin, and related rather to the cellular degeneration than to the interstitial fibrosis.

As to the general frequency of albuminuric retinitis in parenchymatous nephritis, statements vary widely. I should have thought, judging from my own experience, that it was extremely rare in the acute stage, and far from common in the later or chronic stage, and that altogether the frequency was much less than in granular kidney.

Fagge, however, stated that it was far from rare in parenchymatous nephritis; and of Sutton's 50 cases 15 occurred in parenchymatous nephritis, the remainder being in granular kidney. This gives a relative frequency of 15 to 35, or 3 to 7. The actual frequency in relation to the

total number of cases observed was 14 per cent., for tubal nephritis and 9 per cent. only for chronic interstitial nephritis.

I have no actual figures to oppose to these, but my impression certainly does not accord with them.

It is to this acute exudative group that some of the cases of *albuminuric retinitis of pregnancy* belong. The onset is acute, its progress rapid, the impairment of sight early and considerable, and yet in spite of all this the prognosis is by no means so bad either in respect of life or sight.

On the other hand, many more of these cases in pregnancy belong to the degenerative group, and are evidence of granular kidney. In these there is often no complaint of impairment of sight, even when the lesions in the eye are well developed; the course is insidious and chronic; and the prognosis bad in respect of life especially, and of sight, too, if vision be already impaired.

Puerperal eclampsia occurs in either form; in the former rarely without dropsy, and in the latter not infrequently without.

If the view I am expressing prove to be correct, it follows not only that the kind of albuminuric retinitis which prevails in the two forms of renal disease is different, but that it has a different diagnostic value in the two cases. In parenchymatous nephritis the diagnosis of the disease is obvious, and the albuminuric retinitis is an interesting by-phenomenon only. In granular kidney the diagnosis may be uncertain until all doubt is dispelled by the discovery of the characteristic eye-changes. Anyway, in future investigations on the subject it will be well to keep the acute exudative or inflammatory forms (the so-called "extreme" degrees) separate from the more chronic and less obvious forms which are frequently discovered by the ophthalmoscope without any special symptoms suggesting eye lesions.

The Degenerative Form.

The degenerative form of albuminuric retinitis consists in white patches and hæmorrhages. Of these the most characteristic are the white patches.

White Patches.

The white patches are bright spots usually seated in the neighbourhood of the yellow spot, or between the yellow spot and

disc. In their typical and favourite form they are arranged round the yellow spot in a radiating form, like the spokes in a wheel. They are usually found in both eyes, and are often remarkably symmetrical. In very early stages they may be so minute as to be easily overlooked, and may be found here and there only, generally between the disc and the yellow spot. Even then it is remarkable that they are usually present in both eyes, and often symmetrical, though of course in this early stage they are more likely to be unilateral and unsymmetrical.

The white patches may for a long time be the only lesions visible, if we disregard for the present certain changes in the vessels which are possibly still earlier, and will be referred to later.

There are, I believe, two kinds of yellow spot, the one forming bright points *glistening* like fish scales, the other not so glistening or so sharply defined, but *woolly* in appearance. The former are the result of degenerative changes, and their brilliancy depends upon the presence of minutely refractive oil drops, or even cholesterine crystals; the latter are often small patches of exudation only. The former are of slow development and unlikely to disappear at all, or only slowly and after a long time the latter, however, may come and go rapidly.

If there be two kinds of white spots, as I think there are, then the conflicting statements made about them may be reconciled. Thus it is asserted by some authorities that the white patches readily come and go; while others hold that once present they never disappear. The patches of exudation might appear rapidly and also quickly disappear, but the patches of degeneration would persist for long, and possibly till the end of life. Most authorities hold that the brilliant patches are almost pathognomonic of granular kidney, while others state that they are frequently seen in parenchymatous nephritis. If in granular kidney the patches are degenerative and in parenchymatous nephritis due to exudation, this difference of opinion also would be explained.

Of course it may not be always possible to distinguish by the eye alone between these two forms of white spots, yet I think it can generally be done. From a practical point of view the distinction is not so important as it might seem, for in parenchy-

matous nephritis the diagnosis of renal disease is usually obvious, whether there be white spots or not. In granular kidney, however, the diagnosis may be uncertain until the eye-changes are discovered. The white spots, whether they be really exudative or degenerative, have, therefore, in granular kidney a diagnostic value which they do not possess in the other forms of nephritis.

Hæmorrhages.

These usually occur at a later period; they are minute and flame-shaped if seated in the upper layer of the retina, but if seated in the deeper layers are larger and less regular. Sometimes they are numerous round about the yellow spot, but at other times are scattered widely over the fundus.

Similar hæmorrhages may occur in both the exudative and the degenerative forms, though they are probably produced in different ways in each case. In the latter they are the consequences of the vascular degeneration, and due to the rupture of the diseased arteries, in the retina as in other parts of the body; in the former they are probably due to the rupture of over-distended veins, the over-distension being the result of the pinching to which they are subjected as they pass through the swollen disc.

When of considerable size the hæmorrhages naturally interfere seriously with vision, and so may even a small hæmorrhage if seated in the macula. On the other hand, it is surprising how numerous the hæmorrhages may be without any defect of vision at all.

If the hæmorrhage is very profuse, it may burst the retina and fill the eye.

The hæmorrhages are sometimes absorbed completely and leave no trace behind. They rarely lead to choroidal atrophy and pigmentation, as other forms do. Indeed these lesions may follow effusion in parts where there has been no previous hæmorrhage.

There is nothing characteristic about the hæmorrhages in renal disease for exactly similar hæmorrhages may be seen in grave anæmia, as well as in optic neuritis of other origin. Indeed in renal disease some of the smaller hæmorrhages may be connected with the anæmia, and be due to simple extravasation; but the larger ones must be the result of rupture of the vessels from disease.

Detached retina is an interesting, but not altogether rare, phenomenon in granular kidney. It is in most cases due to an effusion of serum beneath the retina, and not, as might be expected, of blood. I have seen hæmorrhagic detachment of the retina, but it is a rare affection. The detachment is usually peripheric—that is to say, occurs at the outer part of the retina, and in very many cases affects both eyes, and is remarkably symmetrical.

Arterial Changes.—Silver-Wire Arteries.

Early as the white patches or degenerative changes are, they are preceded by, and result from, still earlier changes in the vessels.

The lesions in the vessels have been well described by Brailey and Edmunds, and are briefly summed up by them as obliterative arteritis leading to inflammatory degeneration of the structures supplied.

The small arteries and even the capillaries are thickened, degenerate, and rigid. In the arteries the earliest pathological change appears to be a general thickening and hypernucleation of all the coats of the artery without any diminution of their calibre. Gradually the arteries become still more thickened and structureless, and the capillaries hyaline and rigid. Later still the arteries show very great thickening of all the coats, especially the subendothelial part of the intima; this leads to an increased total diameter of the vessel, though the lumen is greatly diminished or may even be completely obliterated. In the most advanced stages some of the arteries may be found in a state of fibroid or structureless degeneration, and similar change may affect many of the capillaries. The changes are exactly the same as those found in the renal vessels.

All the vessels in the retina are affected, but not every vessel, nor even all parts of the same vessel, to the same extent.

The ophthalmoscopic appearances connected with these changes have been admirably described by Marcus Gunn.

The arteries look narrower than usual; they are somewhat irregular in breadth in different parts, sometimes presenting fusiform enlargements, and in others a long tubular enlargement

followed by a constriction; they may be pouched, and thus form small aneurysms; they are often tortuous.

They frequently present a brilliant central streak of metallic lustre (the silver-wire arteries of Gunn). This streak sometimes runs uniformly for some distance along it, at other times it is broken, patchy, or dotty. It does not involve all the arteries, nor even all the branches of the affected artery. It is especially seen in the secondary branches of the central artery, that is to say, at some little distance from the discs.

The whole vessel shows a broader reflex than normal, and gives the impression of unusual fulness and roundness of contour.

The vessel loses its translucency, so that the veins below are not visible through it, or, on the other hand, if the vein lie above, the artery may show through the vein with unusual distinctness.

Where a vein lies below an artery it loses its central streak, showing that it is somewhat pressed upon, and may be distended peripherally.

The whole retina is usually grey and hazy, which Gunn explains on the assumption of there being a small amount of œdema, especially near the macula and in the area of distribution of the most diseased vessels.

The hæmorrhages are frequently due to rupture of the distended veins, but the arteries of course often rupture, too, in the eye as in other parts of the body.

The ophthalmoscopic appearances are connected with the changes in the vessels thus; the narrowness and irregular size of the vessels are explained by the thickening and by the diminished lumen; the tortuosity by the increased rigidity and loss of elasticity; the increased light reflex and the bright central streak by the fibro-hyaline change; the distension of the veins by the great thickening and resistance of the artery as it lies upon them.

The difficulty of circulation leads to the general œdema; the progressive disease of the vessel to hæmorrhage; the defective nutrition of the nerve-cells and fibres to the degenerative white patches. The radiating arrangement of the white patches round the yellow spot Gunn refers to the radiating folds into which the retina in that region is thrown when œdema occurs there.

The silver-wire arteries are not to be confused with the streaks often seen at the side of the vessels, which are either produced

by exudations, as occurs in cases of optic neuritis or in leucocythemia, &c., or by periarterial change, as in the case described by Mules.*

Although the silver-wire arteries are no doubt ophthalmoscopic evidence of the vascular degeneration, as are also the hæmorrhages and white spots, it does not follow that the vessels necessarily show the white silver-wire streaks before the other lesions are visible. On the contrary, I have observed the white streaks develop in the arteries after other lesions had been conspicuous for months.

The changes described, Gunn states, begin to be seen usually between the ages of 40 and 50, but they are not due to old age, *i.e.*, to atheroma, for this does not produce them. They may be seen, however, much earlier in life than this—indeed, at any age, provided the vascular degeneration is far enough advanced.

The changes, of which Gunn gives so good a description, are really the early changes of granular kidney. This his cases prove, for of the 14 cases upon which the paper is based, nine were certainly cases of granular kidney, and three more probably, while in the remaining two only is the diagnosis indefinite.

The dependence of these changes on granular kidney is confirmed by a collateral series of observations which he made. He chose 17 cases of hemiplegia, presumably due to vascular disease, for the purpose of comparing with those in his paper; seven showed characteristic retinal changes such as he had described, and four of these had albumen in the urine; three more had slight vascular changes in the eye, and one of these had albumen; seven had no changes in the eye at all, but one of these also had albumen.

Of these last seven, four died, and *post-mortem* examinations were made. In three of them well-marked granular kidneys were found, and in the other the kidneys were not granular, but as there is no record of any microscopical examination, the absence of interstitial nephritis is not proved.

The papers of Brailey and Edmunds, on the one hand, and of Gunn on the other, are complementary, and together establish the fact that the changes in the eye which we know as albuminuric retinitis certainly depend upon the disease of the vessels, and that at the time these changes become evident, the kidneys are already granular.

They give us no help, however, in determining whether the vascular changes or the renal changes are the primary. On the

* 'Ophthalm. Soc. Trans.,' 1881–82.

whole they seem to show that the vascular changes are late, considering how delicate the retina is and how early the changes in the vessels could be detected by the ophthalmoscope if they were present.

Gunn's paper will fix attention upon the changes in the vessels which precede albuminuric retinitis, and possibly these ophthalmoscopic changes in the vessels will be found at a much earlier period in the disease than is now supposed.

It is a remarkable fact that albuminuric retinitis is almost always bilateral. In the extreme exudative form this is, I believe, always so; but it is also the case usually even when the changes are slight. The changes are not only bilateral, but frequently also remarkably symmetrical, and differ but little on the two sides. In the acute inflammatory form this is not a matter of surprise, for it is probably of toxic origin, and, like other forms of toxic neuritis, is symmetrical. It is more remarkable in the degenerative forms—*i.e.*, in the less acute and chronic—where the changes are more or less mechanical, and consequent on the vascular degeneration.

There are exceptions to this general rule, and the lesions may be well marked in one eye, and absent, either entirely or almost, in the other. Several instances of well-marked *unilateral albuminuric retinitis* have been recorded, and I have recently published two instances myself; but cases of the kind are really rare.

In one of my cases, a male in middle life, the right eye presented the usual features of well-marked albuminuric retinitis of the degenerative kind, with numerous white patches and hæmorrhages; while the left showed nothing except two very tiny white spots, which were so small as to be at first overlooked. In another case repeated examination of the other eye failed to discover any abnormality at all.

Anderson* records a case in which no change in the opposite eye occurred for four years.

It would be interesting to know in what proportion of the cases of granular kidney albuminuric retinitis occurs, but that we cannot yet tell, for we have not the figures upon which any safe conclusion could be based. The figures given vary from 6 to 30 per cent. I should say that in most cases of granular kidney if the patient live long enough albuminuric retinitis does ultimately

* 'Ophthalm. Soc. Trans.,' viii, 130.

develop, but many die from other causes, *e.g.*, from hæmorrhage in the brain, and so do not reach the later stage in which albuminuric retinitis is found.

Other Eye Affections.

The vascular changes of granular kidney are of great importance in relation to various hæmorrhagic affections of the eye. Thus Gunn connects *Hæmorrhagic Retinitis* with granular kidney. Brailey and Edmunds found the retinal arteries extensively diseased in 16 out of 17 eyes excised for *Hæmorrhagic Glaucoma*. Gunn suggests that many cases of so-called *Embolism of the Central Artery* of the retina are really due to thrombosis in an artery so diseased, and that thrombosis here may give rise to retinal change similar to that met with in *Retro-bulbar Neuritis*, which some of the extreme forms of albuminuric retinitis so closely resemble. Similar conditions might be produced by hæmorrhage into the sheath of the optic nerve, of which some instances are recorded.

Cataract is rare in granular kidney, but Gunn suggests that it might be the result of sclerotic change in the ciliary vessels.

Knies records two instances of *iritis*.

I cannot remember to have myself seen any instance of either cataract or iritis which I felt justified in referring to granular kidney, and I should regard their association as accidental.

Extra Ocular Hæmorrhage.

Hæmorrhage occasionally occurs *behind the eyeball* in the orbital chamber. Then sudden pain is felt behind the eye, which is thrust considerably forward. The hæmorrhage may soon extend forwards and become visible beneath the conjunctiva, or, if not, in the course of a day or two the characteristic staining appears in the eyelids or beneath the conjunctiva. In most cases there is no defect of vision and recovery is complete.

Spontaneous hæmorrhage *under the conjunctiva*, just as occurs in aged people as the result of atheroma, is not at all uncommon. In young and middle-aged persons such hæmorrhage is very significant. Similar hæmorrhage *into the eyelids*, especially the upper, is also sometimes met with.

Knies describes one result of hæmorrhage which I mention, but

cannot endorse from my own observation, viz., *paralysis of the ocular muscles*, the consequence of hæmorrhage either into the nerve-trunks or into their nuclei. The cases, he states, recover with or without treatment, but are subject to relapses.

Thus he describes abducens paralysis in a case of albuminuria of 15 years' standing ; this relapsed twice and the patient died.

In another case diagnosed as granular kidney, paralysis of the left troclear nerve occurred, and when, six months later, the patient died, hæmorrhage was also found in the optic nerve.

In a third case there was complete right ophthalmoplegia externa with albuminuria. At first the trochlearis was affected, then the internal rectus, finally the other branches of the third, and later there was unilateral and subsequently bilateral ptosis. All these symptoms ultimately disappeared and the patient recovered.

The obvious objection to all these cases is that third-nerve paralysis generally develops more or less gradually, whereas if it were due to hæmorrhage its onset ought to be abrupt. I cannot recall a case of the kind in which the onset of ocular paralysis was as sudden as the theory of hæmorrhage would require, and I do not think that such unusual cases could well have been overlooked.

Affections of Sight.—Amblyopia.

The early lesions of albuminuric retinitis produce little or no defects of vision, and even in the later stages it is very remarkable how extreme the eye-changes may be without a complaint being made of impairment of sight.

I have recently seen a case with extreme albuminuric retinitis of the exudative form, in which not only could the finest type be read and the finest needle threaded, but the perimeter showed no diminution in the field of vision. I mention this because it has recently been controverted, but of course it is only what is quite familiar in other forms of extreme optic neuritis.

Routine examination with the ophthalmoscope will often discover albuminuric retinitis when there has been nothing to suggest its presence, and will make a diagnosis clear which may till then have been obscure. Indeed the recent progress in our knowledge of granular kidney has been large and coincident with the systematic use of the ophthalmoscope in medicine.

As a rule, we may say that when patients complain of defect of vision, the albuminuric retinitis is already far advanced. When the vision once begins to fail it may fail very rapidly, so that within a week or two it may be completely lost.

Amblyopia.

In albuminuric retinitis, as in optic neuritis, transient attacks of great impairment of sight, or even of complete blindness, occasionally occur, lasting it may be but a few minutes at a time, the sight in the intervals being good and equal to the finest work—they would be called attacks of amblyopia. They may recur during weeks or even months, but, as a rule, vision soon begins to fail, and then is quickly lost.

Amblyopia is often said to be a common symptom of uræmia, and is usually regarded as either of peripheral origin in the retina or of central origin in the brain or special ganglia. Most of these statements date from a time when the ophthalmoscope was not systematically used.

Of toxic retinal amblyopia we know little or nothing, for most of the cases are associated with very definite lesions in the retina visible with the ophthalmoscope.

The amblyopia of central origin, *i.e.*, non-retinal, is often associated with other symptoms, *e.g.*, hemiplegia. In such cases the amblyopia is also in all probability due to lesions of an organic kind. It is not rare when a patient dies of uræmia to find the symptoms which appeared to be merely toxic or functional explained by an organic lesion in the brain, viz., small hæmorrhages, and in cases of so-called uræmic amblyopia in which the defect of sight persists after the uræmic attack is past we must presume that there has been an organic lesion.

In cases of toxic amblyopia recovery ought to take place in the course of a few hours or less, though instances are recorded in which recovery has been sudden and complete even after four days.

Some clue as to place of origin of the amblyopia may be given by the condition of the pupil. If the pupil-reaction be retained and normal, the lesion, if there be one, must be high up or cortical.

If the reaction to light be lost there is probably some affection of the optic nerve or optic ganglia. In some cases described by Lytton, in which absence of the pupil-reaction to light occurred, each attack was associated with pronounced swelling of the discs, so that there was probably some hæmorrhage or lesion in the optic nerve.

Toxic amblyopia no doubt occurs, and may even be ununi-

lateral, as uræmic fits may be; yet lesions are now found much more frequently than used to be thought likely.

Prognosis of Albuminuric Retinitis.

The significance of albuminuric retinitis is grave in respect of life even more than in respect of sight.

So far as the eye-changes are concerned, something will depend upon their nature and upon their cause. Thus hæmorrhage may be absorbed and disappear whether due to granular kidney or not. White patches which are due to exudation may also disappear, but the white patches which are due to degeneration, such as are usually met with in granular kidney, I have not myself ever observed to disappear. Again, where the albuminuric retinitis occurs in the course of chronic parenchymatous nephritis it is possible that if the primary disease gets well the eye-changes may also disappear and recovery of health and sight be complete.

1. *The Exudative Form.*—Paradoxical as it may seem, it is in the so-called extreme forms of albuminuric retinitis or, as I should call them, the exudative forms that the prognosis is, both in respect of life and in respect of sight, not nearly so grave as it might appear to be, for if the kidney mischief recover the eye lesion may resolve and the sight be completely restored.

In respect of sight, indeed, the prognosis is better with albuminuric retinitis of this type than it is with similar forms of neuro-retinitis due to other causes. Possibly this is due to the fact that in albuminuric retinitis the exudation may be more of the nature of œdema or dropsy than of inflammation, so that its effects are rather mechanical than degenerative. Anyway many of the cases recover without any defect of sight.

If, as in pregnancy, the cause return, the retinitis may also return, and with each succeeding pregnancy the prognosis in respect of sight of course becomes worse, but the significance of the eye-lesions is usually dwarfed by the importance of the renal disease of which they are the accompaniment, for the danger of renal disease during pregnancy is well recognised.

In respect of life the prognosis is that of the renal disease, and all that I think the albuminuric retinitis in these cases does is to show that we have a form of parenchymatous nephritis to deal with of a somewhat unusual severity.

Thus Sutton's figures show that while in cases of chronic parenchymatous nephritis without retinal change 22 per cent. only were fatal, in those in which retinal changes were present no less than 60 per cent. died. In 10 fatal cases the average duration after the retinitis was first noticed was four months, and from the time of the beginning of renal symptoms only 11 months.

2. *The Degenerative Form.*—The degenerative forms of albuminuric retinitis have great importance in granular kidney both in respect of sight and in respect of life.

In respect of sight.—The white patches of granular kidney rarely disappear; I have not myself seen any instance of their disappearance, but they are very often present without much defect of sight. The longer they persist the greater the chance of impairment of sight developing, either as the result of progressive degeneration of hæmorrhage or of other changes in the eye already referred to. When once such impairment of sight has commenced it steadily progresses. Sight is, however, rarely seriously impaired, still less completely lost, until the case is near its end, unless of course there has been detachment of retina or severe hæmorrhage.

The ophthalmoscopic changes in the vessels are important as evidence of an arterial degeneration which is not limited to the eye, and it is a visual evidence of the risks to which the patients are subject from the disease of the vessels elsewhere, *e.g.*, in the brain. But, as I have stated in most of these cases, the eye-changes are but rarely seen until the other signs of granular kidney are obvious enough.

In respect of life.—The duration of albuminuric retinitis, or of life after its appearance, it is not possible to determine accurately, for albuminuric retinitis is not as a rule discovered in its early stages, either because the patients do not come under observation early enough, or because the eye is not examined with the ophthalmoscope until defects of vision are complained of.

In granular kidney, speaking generally, the duration of life after albuminuric retinitis has been discovered is short.

Thus, Miley traced 45 cases, and found the average duration of life to be under four months from the time when the eye-changes were first observed, and though one of the patients lived 18 months and two 14 months, all the rest died within the year. In nine cases in which the first appearance of eye-changes was noticed the duration averaged less than six months, and in two cases it took a month for white patches to develop after the appearance of hæmorrhages.

Bull, in 100 cases of albuminuric retinitis, found that 86 of 103 died,

57 in the first year, 12 during the second, and 17 after a longer period. Of the 17 remaining cases that were still living at the time his paper was published, 14 had been seen only during the six months immediately before the paper was written, but one had been diagnosed seven years previously.

Where the patients are in a condition to take the best care of themselves life may be prolonged for some time.

Thus, the Baroness Possauer found that within two years of the time of observation, among hospital patients, all the men died and 68 per cent. of the women ; among private patients, of the men 59 per cent. only, and of the women 53 per cent.

Instances, however, are recorded of considerable duration.

Thus, I have a patient of my own who has been under observation now for more than four years, and she is still in much the same general condition as she was when she was first seen ; and I have already referred to a case of Miley's in which the patient was known to have lived for more than seven years.

Anderson records some remarkable cases. One died 12 years after the albuminuric retinitis had been detected, and two others were living four and seven years afterwards respectively.

Cases such as these are quite exceptional, and speaking generally, when patients with granular kidney are found to have albuminuric retinitis they have not many months to live.

I may bring this part of my subject to a conclusion by summing up the facts which, I think, justify the drawing of a sharp distinction between the two forms of albuminuric retinitis, the degenerative and the exudative.

They stand in strong contrast with each other in the following respects :—

(1) Of the form of disease with which they are associated—the degenerative with granular kidney, the exudative especially with parenchymatous nephritis.

(2) Of their nature and cause—the exudative being inflammatory and probably toxic in origin, the degenerative consequent on the vascular changes, and more or less mechanical in origin.

(3) Of sight—for with the exudative, even in the extreme forms, recovery may take place with little or no defect of sight, but with the degenerative the impairment of sight, if there be any, is usually progressive.

(4) Of diagnostic value—the exudative being an interesting bye-phenomenon of chronic parenchymatous nephritis, the existence of which is obvious enough, while in granular kidney the degene-

rative often makes the diagnosis certain in cases which have been hitherto obscure.

(5) Of life—for while in both cases it indicates a grave form of renal disease which may of itself prove fatal, in granular kidney it indicates in addition all those dangers to which arterial disease exposes the patient.

I think, therefore, that the distinction is not only justified by the facts but explains many of the apparent contradictions which are made by different writers.

LECTURE III.

Symptoms.

Granular kidney may exist for a long time without appearing to affect the health or producing symptoms—when symptoms appear, no matter what their nature, the disease is always far advanced, and in its later stages.

The symptoms are multifarious and, unless there be intercurrent nephritis, in no way of themselves suggest renal disease; so that they are very misleading, and the true nature of the case is often overlooked.

They fall into two groups, the cardio-vascular and the toxæmic.

I call the latter toxæmic rather than renal, because the symptoms, though no doubt of renal origin, are not such as are commonly understood by the name renal and do not of themselves point to the kidney as their cause.

Speaking generally, the cardio-vascular symptoms occur first and the toxæmic subsequently, though they may both be present together.

The cardio-vascular are important, because they often cause death earlier than might otherwise occur.

The toxæmic always develop if the patient live long enough. They depend upon the wasting of the kidney, and become manifest when it has reached a certain degree and progress with it, *pari passu*.

A.—Cardio-Vascular Symptoms.

The cardio-vascular symptoms are more or less mechanical or accidental. The cardiac are summed up in heart failure. The vascular fall into two groups: the first is formed by hæmorrhage and its results; the second by chronic degenerative affections, especially in the nervous system, the result of imperfect nutrition through the diseased vessels.

I. Cardiac.

Heart-failure is very often the first symptom which causes anxiety. Thus a little shortness of breath and palpitation on exertion are complained of, with, it may be, slight cardiac pain and swelling of the feet. The heart is found hypertrophied or dilated, and without obvious cause in the heart itself.

As in cardiac-failure of other origin, the symptoms are usually gradual in onset, but they may come on suddenly after violent effort, or may follow a serious illness.

The pain is usually slight, but may be severe enough to be called angina.

The cardiac symptoms may take the form of repeated attacks of severe dyspnœa.

Œdema may be extreme. This is in most cases in great part of cardiac origin, but where ascites predominates it may suggest the combination of cirrhosis of the liver with granular kidney.

Where the heart is affected with valvular disease as well, symptoms of cardiac failure are of course more likely to occur.

In elderly persons these symptoms might be the result of atheroma, but in middle life and in the young granular kidney is the most likely cause, and, attention being thus directed to the kidney, the signs of granular kidney are discovered. Yet even in the elderly there may be but little atheroma, and the cardiac failure be, as in younger persons, the result of granular kidney.

Cardiac symptoms are, however, not constant, even where the heart is greatly hypertrophied or actually diseased.

I have given an account of a young man who was under my observation for some years with aortic regurgitation. He developed granular kidney and subsequently died of uræmia, yet his heart gave him no trouble throughout, and his symptoms were of another kind entirely.

In this connection I may mention Cheyne Stokes breathing, which in granular kidney is almost without exception of cardiac

origin. Although described as occurring in uræmia it is hardly ever seen except in those cases in which there is heart failure, to which it is really due. It is, as in many other forms of heart affection, of grave omen, and may usher in the end, which is rarely long deferred.

Acute pericarditis belongs to a somewhat different category of symptoms, but this is perhaps the most convenient place to refer to it. It is always a very severe complication, and even where it does not itself cause death it is a warning that the end is near.

In the course of a case already recognised as granular kidney it may produce no symptoms, and be detected only by physical signs. It is then liable to be overlooked or discovered only accidentally.

I was asked to see a gentleman in consultation because he had suddenly developed pericarditis without any obvious cause. I found the patient in bed, well nourished and muscular, with a somewhat sallow, unhealthy complexion, looking not seriously ill. There was well-marked pericarditis, friction being unusually loud. His breath was slightly short, and his pulse a little hurried, but except for feeling generally ill he seemed to have nothing special to complain of. The urine contained a small amount of albumen, the arteries were thickened, and the tension raised.

The patient had been in perfect health until three weeks before I saw him. He had been hunting two or three times a week, and it was because he found he could not see the wire fences that he began to be uneasy about his eyesight, and this on one occasion caused him to get a fall. The eyesight gradually got worse, and it was this only which took him to the doctor. A week or so before I saw him his vision had become so considerably affected that he could not read at all; the pericarditis was discovered by accident, and he was then sent to bed, much against his will. Ophthalmoscopic examination showed the most extensive albuminuric retinitis, with numerous white patches in both eyes, and a few hæmorrhages; the discs were greatly swollen, so that the condition fully explained the loss of sight. The breath was somewhat urinous; the quantity of urine passed was sufficient in amount, but the specific gravity was low. A very grave prognosis was given, and in the course of a few days the patient died.

Now this patient had thought himself to be in perfect health, and was out hunting a month only before his death.

II. The Vascular.

The vascular lesions produce three sets of symptoms :—

(1) Those due to rupture and consequent hæmorrhage.

(2) Those which result from nutritive disturbances in the parts supplied by the diseased vessels. This is especially met with in the central nervous system and in the eye.

(3) Besides these it is necessary to refer to aneurysm, and that not only of the small vessels, *e.g.*, one of the arteries of the base of the brain, but of the main trunks, such as the thoracic aorta.

(1) *Aneurysm.*

The connection of granular kidney and aneurysm of large vessels is, I believe, a new point, but it is clearly shown in the *post-mortem* statistics from St. Bartholomew's Hospital (Woodbridge), for out of 219 cases of granular kidney, the rupture of a thoracic aneurysm was the cause of death in 33, *i.e.*, in 6 per cent.

In the same series there were, moreover, two cases of death from the rupture of small aneurysms at the base of the brain.

There is no reason why, if a vessel is diseased enough to rupture, it should not first lead to an aneurysm.

To the possible causes of aneurysm, viz., laborious work, drink, and syphilis, must now be added granular kidney.

(2) *Hæmorrhage.*

The affection of the vessels being general, the hæmorrhage may take place in almost any part of the body.

Cerebral Hæmorrhage.—It is in the brain that its most serious effects are produced, and *post-mortem* statistics prove the remarkable frequency with which granular kidney is found associated with cerebral hæmorrhage.

Dickinson states that 41 per cent. of all cases of cerebral hæmorrhage were due to granular kidney, and Pye-Smith states that in 75 per cent. of cases of granular kidney death is brought about by cerebral hæmorrhage. On the other hand the statistics of St. Bartholomew's Hospital only show about 15 per cent.

In elderly persons, of course, due allowance must be made for atheroma; but even elderly persons may have granular kidney and little atheroma; while in middle life granular kidney is the usual cause, and in the young may explain cases of cerebral hæmorrhage which would otherwise present great difficulty.

Thus among Woodbridge's cases there was one at the age of 19, one at 27, one at 30, and two at 37.

Of course many patients with cerebral hæmorrhage do not die of it, but are left with paralysis, as in the following case :—

A young man, aged 28, recently presented himself with left hemiplegia. The attack had come on suddenly in the midst of apparent health, and was evidently due to hæmorrhage. No cause was obvious until the thickened radial and high arterial tension suggested granular kidney, and albumen was found in the urine.

The difficulties of diagnosis are sometimes considerable. In the following case the symptoms at first seemed to suggest fracture of the skull :—

A man of about 40 was brought into the hospital unconscious and bleeding from a cut on the head. The question was raised as to whether he had a fractured skull, but the symptoms seemed to fit in best with uræmic coma, and that diagnosis was made. The patient died within 24 hours, and on the autopsy no lesion was discovered except granular kidney. The brain was free from hæmorrhage.

Where the patient dies of apoplexy *post-mortem* examination may show the centre of the brain ploughed up by a large hæmorrhage of the common kind; but sometimes, without any extensive laceration of the brain, a large number of minute hæmorrhages are found of that peculiar kind known as miliary aneurysms.

Where the small hæmorrhages are not numerous they may produce no pressure signs, but only localised symptoms of peculiar character depending on their position. They probably explain some cases of unilateral fits occasionally observed in uræmia. Even when death follows uræmic fits of apparently the ordinary kind, multiple minute hæmorrhages are not unfrequently found, so that the distinction between the hæmorrhagic and the toxic form of uræmia is not always possible.

Bulbar Hæmorrhage.—In the medulla a very small hæmorrhage of this kind may lead to death, or to localised nuclear paralyses of special nerves. Pontine hæmorrhage is, of course, rare, but in some instances granular kidney is the cause of it.

Spinal Hæmorrhage.—Of hæmorrhage into the spinal cord below the medulla I have met with no example myself in granular kidney, nor do I know of any conclusive case recorded.

Ophthalmic Hæmorrhage.—The various forms of hæmorrhage in connection with the eye are of great importance, but have been already dealt with.

Of other hæmorrhages which are not of such immediately serious character as the preceding, epistaxis and hæmaturia are the most interesting and important.

Epistaxis.—Epistaxis is common and may be the first symptom of disease, so that its significance may be missed.

Even in the young granular kidney must not be disregarded as a cause of repeated epistaxis.

Hæmaturia.—The next most important group of cases is that in which blood is found in the urine. Attention had not been drawn, so far as I know, to granular kidney as a cause of hæmaturia until I published some cases a few years ago, but the fact is now generally recognised.

The blood is intimately mixed with the urine, and is usually bright in colour, and may look almost like pure blood, but even then there are no clots. I suggested, in my original paper, that the hæmorrhage was probably vesical in origin, as the urine was, in the absence of acute nephritis, rarely smoky or dark in colour. This may be correct in some cases, but two of Bowlby's cases show that the blood may also come from the kidneys themselves, and it has been seen during life with the cystoscope passing out of the ureters. This form of hæmaturia has led to many mistakes, chiefly to the diagnosis of calculus in the bladder —or in the kidney if there has been lumbar pain or attacks of renal colic. Operation has even been suggested and performed, and in fatal cases the absence of any lesion except granular kidney has been proved by autopsy.

In my original paper I refer to a case communicated to me by Dr. Sharkey, in which the patient, a young girl, passed so much blood with the urine that the bladder was sounded and, failing to find a stone, dilatation of the urethra and digital exploration of the bladder was suggested. To this Dr. Sharkey did not consent, and, the patient dying shortly after, no stone was found *post-mortem*, but only markedly granular kidneys.

Copious hæmorrhage, as in the cases described, is not common, but hæmaturia in slight amounts is frequent enough, and it is often long continued or recurrent. To this recurrent slight hæmaturia experience has led me to attach considerable diagnostic value.

Hæmatemesis.—Hæmatemesis is very rare, but I have seen one instance of it.

The patient was a man of 45 years of age, fairly well nourished, but a little pale; a cab-driver, but a total abstainer for the last 13 years. The arteries were greatly thickened, the tension high, and the heart slightly hypertrophied. At times there was a small amount of albumen in the urine, but it was often absent. Four years ago the patient had an attack of profuse hæmatemesis, and brought up one or two pints of blood. He had no definite gastric symptoms of any kind, and nothing whatever pointing to the presence of ulcer then or subsequently. This attack left him for a time very weak, but he recovered and went on well till a few weeks ago, when he had another attack of hæmatemesis, bringing up also a considerable amount of blood. A fortnight ago he had another attack, bringing up about a pint of blood, and it was this which brought him to the hospital. The case was obviously one of granular kidney, though there was no albuminuric retinitis to make the diagnosis incontrovertible. The only other question was whether anything of the nature of gastric ulceration existed. All that I can say is that there was no sign and no history of it. This, of course, is not quite conclusive, for the signs of gastric ulcer may be latent. There was, at any rate, no evidence whatever of cirrhosis of the liver, and the history of total abstinence for so many years was against it.

Hæmorrhage from the Bowels.—Hæmorrhage from the bowels is very rare, and in this connection it is interesting to remember that in a small number of cases ulcers of the intestine have been discovered in granular kidney, of which no signs had been given during life to suggest their presence. In other cases, however, the autopsy has shown no gross lesion to explain the hæmorrhage.

Hæmoptysis.—Hæmoptysis in the course of granular kidney, though I can have no doubt it occurs, is an event I have not personally met with. Hæmoptysis of this kind probably stands in the same relation to granular kidney as the hæmoptysis of old people does to atheroma. Sir Andrew Clarke described these cases as Arthritic Hæmoptysis. The name, though striking, is really one to which much objection can be taken, though it expresses an important clinical fact, viz., the old people may suffer from hæmoptysis, even to a considerable amount, as the result of atheromatous disease of their vessels, this change being in Sir Andrew Clarke's opinion the consequences of gout. The condition is, at any rate, a rare one, for Sir Andrew Clarke had only met with 15 instances of it in the course of 20 years' experience.

Metrorrhagia.—I have recently had to deal with a case in which very troublesome and persistent hæmorrhage occurred from the uterus, which was ultimately, after some difficulty, checked by

full doses of ergot. There was no diagnostic difficulty in this case, for the signs of granular kidney were already obvious enough.

Oozing-Hæmophilia.—In the last stages of granular kidney the patient may pass into an almost hæmophilic condition, in which slight, though continuous and almost uncontrollable, oozing takes place from various parts of the body, from the gums, nose, tongue, lips, pharynx, vagina, or from any wound or scratch in the skin. A considerable amount of blood may be thus lost, and the anæmia from which the patient is already suffering be very greatly aggravated.

Purpura.—Although almost any part of the body may be the seat of hæmorrhage, it is remarkable that while acute and severe affections of the skin are not rare in granular kidney, they show no special hæmorrhagic tendency, and it is quite rare for any of these eruptions to be of such a character as to be fairly called purpuric.

(3) *Degenerative Changes.*

The next group of cases is an important one, viz., that in which, as a consequence of arterial disease, degenerative changes result in various tissues; the most delicate of these is the nervous system.

To the connection between granular kidney and some chronic affections of the nervous system Gull and Sutton long ago drew attention, but this subject has not of recent years received the attention it deserves, perhaps because Gull and Sutton made their theory of a primary arterio-capillary fibrosis depend too much upon it.

The changes in the eye are the most striking and most familiar, but these have been already dealt with.

Bond has drawn attention to the remarkable frequency of granular kidney in *post-mortem* examinations of the insane, and has shown that it is nearly double that of a general hospital like St. Bartholomew's. He rightly concludes that the two diseases stand in more than an accidental relation to one another.

Although some of the diseases of the nervous system associated with granular kidney may rightly be attributed to defective nutrition in consequence of the disease of the vessels, it must not be forgotten that they may also be toxic in origin.

B.—Toxæmic Symptoms.

Acute and Chronic Uræmia.

The toxæmic symptoms fall into two groups: in the one they are of acute onset and great severity, and usually lead rapidly to death. In the other they are of more gradual onset, of longer duration, of less apparent severity, and of a very indefinite character. They are respectively called *acute and chronic urœmia;* but as the groups stand in strong contrast with one another it would be well if different terms were employed to denote them. For the acute cases uræmia in its ordinary acceptation might be retained. For the chronic group on account of its varied and indefinite symptoms chronic renal toxæmia would be the better term.

There are three main theories of uræmia, which may be termed the "retention" theory, the "perverted metabolism" theory, and the defective "internal-secretion" theory.

The general cachexia which develops in some forms of granular kidney bears a striking and suggestive resemblance to the condition which is met with in Addison's disease or even in myxœdema, but there is no definite proof so far of an internal secretion in the kidney.

The retention theory, which maintains that the symptoms are produced by the retention of some of the ordinary constituents of the urine in the body, such as urea, creatin, creatinin, &c., is now discredited.

The third theory only remains, viz., that which refers the symptoms only to perverted metabolism. According to this theory the products of metabolism—extractives as they are usually called—are either produced in abnormal amount, or not properly elaborated and eliminated in the normal way. In either case they would be increased in the tissues and this increase can be demonstrated. The symptoms of uræmia might thus be referred to "extractive" poisoning, or, as it is sometimes called, "auto-intoxication."

Various poisons are normally produced in the tissues which ought to be eliminated by one organ or another, and especially by the kidneys. If their elimination be suspended or inadequate, toxæmia results. A healthy man, it is said, manufactures in little more than two days enough poison to kill himself.

There are many different extractives and they have different physiological actions; thus the symptoms produced might vary according as one or other of the extractives predominated.

Bouchard has separated from the urine no less than six different toxic substances, which fall into two groups, of which the one produces somnolence, coma, and salivation, and the other myosis, fall of temperature, and convulsions.

This is Bouchard's theory of multiple intoxication. According to this theory the poisons are produced in the body, and by processes not differing essentially from those which take place in health. The kidney plays its part only so far as it makes the elimination of the poisons defective; and Uræmia would thus be brought into close relation with other conditions in which the normal metabolism of the body is perverted.

Clinically the symptoms of uræmia are closely simulated by many conditions which at first sight appear to have nothing to do with one another; thus the typhoid state which develops in many fevers is like some forms of uræmia, and Murchison did not hesitate to call it uræmic. Again, when the tissues are rapidly wasting, as in malignant disease, symptoms arise towards the end which, if they happened in a case of renal disease, would be called uræmic; so, again, in the later stages of cirrhosis hepatis, diabetes mellitus, the last stage of Addison's disease, of myxœdema, and indeed in almost all slowly-dying persons.

We might thus place all these conditions together under the one heading of Toxæmia, adding some prefix, as renal, hepatic, supra-renal, thyroid, diabetic, febrile, or others to indicate what is believed to be the cause in the particular case.

Symptoms resembling uræmia may thus arise under conditions with which the kidneys may have nothing to do. When, as in uræmia, the kidneys play the chief part, it is very tempting to refer the symptoms to defective internal secretion, for it is certainly not due simply to defective elimination.

It is for this reason that I prefer to call Uræmia *Acute and Chronic Renal Toxæmia*, so as to bring it into its proper relation with other allied clinical conditions.

Uræmia may thus arise in two ways:—

(1) The metabolism may be normal and the elimination defective.

(2) The metabolism may be increased or perverted, while the elimination is not deficient and may be, on the contrary, much increased.

In uræmia the tissues are enormously rich in extractives, and that even when elimination by the kidney is active; when elimination by the kidney is impaired the extractives are still further increased. This is not inconsistent with the view that the increased metabolism of the tissue itself may be the result of a defective internal secretion by the kidney.

If, then, we are thus led to associate the symptoms of chronic uræmia with chronic extractive poisoning, *i.e.*, with the presence of abnormal amounts of extractives in the tissues and the blood, we are tempted to refer the acute symptoms which often develop with so little warning to defective elimination by the kidney, and we might thus associate acute uræmia with the condition of the kidney-cells in granular kidney just as we do in parenchymatous nephritis.

Chronic Renal Toxæmia.—Chronic Uræmia.

It is to the chronic toxæmic group that many of the symptoms belong which bring the patient suffering with granular kidney under medical observation. They may be of such a kind as to indicate almost any part of the body as the seat of disease rather than the kidney, and may thus lead to frequent errors in diagnosis. This is especially the case in connection with the digestive and nervous systems. As the disease advances the general nutrition becomes profoundly affected, and a cachexia may develop which may be as extreme as that of malignant disease, or of Addison's disease.

(1) *Gastro-Intestinal Symptoms.*

Dyspepsia.—The gastro-intestinal symptoms are often pronounced and may be very misleading.

Obstinate dyspepsia, especially if associated with pain, might suggest ulcer of the stomach, and this might be attributed, if there were much cachexia, to malignant disease, a suspicion which would be strengthened if hæmatemesis also occurred. As a matter of fact, hæmatemesis is rare, for the hæmorrhage is not likely to be profuse, and unless it were profuse the blood would not be vomited.

Vomiting.—Vomiting is often a source of difficulty, for it is very obstinate and does not stand in relation to the taking of food.

When to the vomiting optic neuritis is added, confusion with intracranial disease, such as cerebral tumour, abscess, or meningitis, is only to be expected. The vomiting may occur only or chiefly in the morning, and might then suggest pregnancy, or potus. I have seen cases of granular kidney confused with all these conditions.

Diarrhœa.—The symptoms in connection with the bowels are not, as a rule, so puzzling. Diarrhœa is common with any form of kidney disease, but there are cases in which diarrhœa, extremely obstinate or almost uncontrollable, is practically the only symptom.

Cramps.—The dyspeptic symptoms are often associated with cramps. I have seen several cases in which cramps were the chief complaint.

(2) *Gout.*

Cramp naturally leads to the consideration of gout.

The occurrence of chronic kidney mischief as the result of long-standing gout is well recognised; but in many cases it is quite possible that the opposite relation exists, viz., that the patients are gouty because their kidneys are granular.

Indeed it has been stated that no young person has gout unless the kidneys are granular. This I believe to be an over-statement, but it represents a part of the truth. I do not know how far the uratic deposit in the joints can be taken as proof conclusive of gout, because it may be found in patients who have never presented during life any gouty symptoms. It is, however, certainly true that a very large number of cases of granular kidney have such deposit in their joints, though this may really not be more than an evidence of the renal inadequacy.

The two affections may certainly coexist side by side for years, but it is not easy to determine the true relation in which they stand to each other.

Per contra, gout is not a necessary consequence of granular kidney; in other words, patients may have granular kidney for years and die of it without ever having suffered from gout at all, or the gout may develop quite late in the course of the disease.

(3) *Lead Poisoning.*

Gout in the course of granular kidney naturally suggests lead poisoning, for the three affections—gout, granular kidney, and lead poisoning—stand in close relation to one another.

The close association of chronic lead poisoning and granular

kidney is interesting as supporting the view that granular kidney is the result of the circulation of some toxic substance in the blood, which, like lead, excites cirrhosis in the kidney, just as alcohol produces cirrhosis in the liver. On the other hand it is also possible, considering that all persons who work with lead do not develop granular kidney, that the opposite relation may exist, viz., that they suffer so much from lead symptoms just as others do from gout because their kidneys are granular already.

(4) *Cachexia.*

Patients with granular kidney may for a long time preserve their normal appearance of health, and that even when the signs of granular kidney are well marked; but as a rule as the kidney disease advances the nutrition suffers greatly. The complexion loses the transparency of health, and becomes thick, muddy, earthy. The patients become feeble and anæmic, lose health and strength, and often flesh.

Failure of health and strength in persons of middle life, without any evidence of malignant disease, should always excite the suspicion of granular kidney.

The cachexia stands in direct relation to the kidney wasting, and, when it is extreme, is an indication that the destruction of the kidney substance has approached dangerously near to the limits compatible with life.

The cachexia of granular kidney is characterised by anæmia and asthenia, and to some extent by loss of flesh, but emaciation is rarely carried to that degree which is met with in malignant disease.

It is often out of all proportion to the other symptoms, or may even exist alone. In some cases the anæmia predominates, and in others the asthenia. Thus, I have seen instances in young people in which chlorosis has been diagnosed.

In those cases in which extreme anæmia is the chief symptom I do not know that the blood has been systematically examined. In the few instances in which I have had such an examination made the changes have been those of simple but extreme anæmia.

The asthenia is often extreme, so that the patient can hardly stand or walk without assistance.

Of this extreme asthenia I have seen two remarkable instances recently. One of these was under treatment for a skin affection and will be referred to in another place. In this case, with rest and feeding, the

asthenia greatly improved for a time, but recurred a few months later. In both cases the asthenia was ultimately fatal.

The second case, a woman aged 41, first came under observation 12 months previously for general pityriasis. She had some albumen in the urine and was so extremely feeble that I did not expect her to live. After a few days' stay in the hospital she improved a little, and insisted on going home as I thought to die. However, she lived on, and in the course of time lost the skin eruption. About 10 months later she appeared again, more anæmic and ill than before. She was again admitted into the hospital, and died two or three days later. I never saw a patient so feeble and cachectic with so little to show for it; she was like a patient dying in the last stage of malignant disease or of Addison's disease. I examined her with the greatest possible care to discover any other cause than granular kidney, but failed to find one. Even the diagnosis of granular kidney at that stage was difficult, for the tension was extremely low and the artery but slightly thickened; there was a small amount of albumen in the urine, out not more than might have been due to her feeble condition, and the eyes showed nothing.

When the *post-mortem* was made all the organs were carefully examined and absolutely nothing found except granular kidney. The kidneys weighed only 4 ounces together.

The only history I could obtain was that the patient had been ailing in an indefinite way for about six years but had been in fair health until the skin eruption appeared three weeks before she was first seen. From the time when the skin eruption disappeared there had been no other symptoms except those of extreme anæmia and asthenia.

The cachexia was so extreme that many to whom I showed the case thought that granular kidney could not be sufficient to explain it, and that there must be some malignant disease which could not be detected. The autopsy, however, confirmed the clinical diagnosis made.

(5) *Skin Affections in Renal Disease.*

This subject has been but little written upon, and yet it is not without its clinical importance. The cases fall into two groups according as there is general œdema or not.

Where the subcutaneous tissue is distended with œdema the nutrition of the skin will naturally suffer, and from very slight local causes rashes are likely to be produced. Where there is no œdema the rashes are probably of a toxic origin.

(1) Rashes Associated with Œdema.

These are for the most part erythematous in nature, transitory in duration, produce but few symptoms, and when localised have but little clinical importance. If widespread the prognosis is less favourable, and becomes the more unfavourable the more general the eruption is.

Patches of erythema are often found on the inside and front of the thighs, and on various parts of the trunk, especially the

lower part of the abdomen. As a rule they last but a few days, and then do not itch or burn or produce any discomfort.

Sometimes the rash is of a papular lichenous character, and is then generally met with on the backs of the thighs and arms.

Eczematous eruptions are not at all uncommon where there is much œdema in places where the parts lie in contact with each other.

If the skin is leaking, as after puncture or when blebs have formed and burst, various forms of rash occur; these are often eczematous, and produced by the irritation of the discharge, or erythematous, and spread in a way which closely resembles erysipelas or other infective eruptions, and are no doubt of septic origin.

As in diabetes so in dropsy the tissues lose their resistance to pathogenic organisms, so that if infection occur it will produce very grave effects. This used to be so far recognised that puncture or drainage of œdematous limbs was regarded as a very dangerous proceeding from the liability to phlegmonous inflammation, or even gangrene. Under the use of strict antiseptic precautions this danger has almost entirely disappeared.

(2) Rashes without Œdema.

These occur almost exclusively in granular kidney. Generally widespread, or even universal, they are of great obstinacy and of grave significance. Localised eruptions similar to those already described occur also in granular kidney, but these are not of much importance.

The general rashes vary in character. They may be described as :—

(1) Erythema.
(2) Pityriasis.
(3) Dermatitis exfoliativa.
(4) General eczema.
(5) A discrete, papular eruption, sometimes lichenous, sometimes resembling chronic urticaria.

I have met with instances of each. In the few cases in which I have seen the initial stage, the rash has commenced as a localised, patchy erythema on the lower part of the abdomen, or on the inner and front parts of the thighs.

It is strange, considering the frequency with which hæmorrhage

occurs in connection with granular kidney, that hæmorrhagic eruptions are so rare. I have seen one or two instances of small hæmorrhages into the skin (petechiæ), usually on the legs. They were probably due to failure of the heart, and mechanical in origin, but I do not remember ever to have had a case under my own observation which could be fairly called purpura, nor, as a rule, have the skin eruptions of granular kidney a specially hæmorrhagic character.

Dr. Colcott Fox* has recently described a case of hæmorrhagic erythema in the course of granular kidney, in a woman of 61. The rash began on the face on January 5th as an erythema, with a few petechiæ on the legs. Five days later (January 10th) it became general, and looked then like severe purpura. Purpuric spots occurred on the gums and palate ; on January 15th retinal hæmorrhage was found, and on the 16th melæna occurred ; on January 17th the patient died.

The only lesion found on the autopsy was in the kidneys, which were small, red, and granular. No plugging of the cutaneous vessels was found, nor any micro-organism in the hæmorrhagic spots.

Skin eruptions, of whatever kind they are, occur only late in the disease and when the symptoms are well marked, so that the diagnosis of granular kidney is usually obvious, and this is perhaps the reason why they have been so long overlooked, or at any rate not described. In some cases, where the diagnosis of granular kidney is not so obvious, the patients come under treatment for the skin affection, and the essential disease is frequently missed; or, if albuminuria is discovered, it is regarded as the consequence of the affection of the skin, and its significance missed.

The association of a generalised skin eruption with albuminuria is of great importance, and justifies a more cautious prognosis than might otherwise be given. Evidence of granular kidney is often found if looked for, and then I believe the prognosis is very grave and the end not far off.

(6) *The Nervous System.*

It is in connection with the nervous system that some of the most interesting conditions arise.

Headache, Giddiness, and Vomiting.—These symptoms are sometimes the first to cause the patient to seek advice, and when they are associated with optic neuritis the resemblance to cerebral tumour becomes very close.

* 'Clin. Soc. Trans.,' 1899.

The vomiting is often of cerebral or nervous origin, for it stands in no relation with food, and is often periodic.

Headache is a very common and prominent early symptom; it is often of a neuralgic character, occurring in the most intense paroxysms, and is occasionally diagnosed as hemicrania—that is to say, that its association with granular kidney is overlooked.

Respiratory Disturbances (Renal Asthma).—The respirations in uræmic states vary much. Thus they may be very slow; I have seen them as low as 10 in the minute, but they are deep, and there is no dyspnœa. Or they may be rapid, noisy, and panting, not unlike what is sometimes seen in diabetes; in this form, again, there is no true dyspnœa.

Cheyne Stokes breathing is usually of cardiac origin.

Dyspnœa, even if paroxysmal, is often cardiac; and if not cardiac, is frequently due to emphysema and its attendant bronchitis, but the bronchitis itself may be of toxic origin.

Any attack of the nature of true spasmodic asthma is very rare, and I must express myself sceptical as to its existence; at any rate, in my own experience I have certainly never met with a conclusive case.

Peripheral Neuritis.—Peripheral neuritis is a rare condition in granular kidney, but the following was, I think, an instance of it:—

A young man, aged 27, came under my care with the signs of peripheral nephritis, both his arms and legs being affected. Shortly afterwards he developed œdema and a good deal of albuminuria, the symptoms being apparently those of acute nephritis. He recovered from both his peripheral neuritis and most of his symptoms of nephritis, but he was left with a small amount of albumen. His arteries were a little thick, and during his convalescence he developed slight optic neuritis; but this passed off, and he was dismissed from the hospital fairly well, except for the small amount of albumen present. He remained, as it was thought, in fair health until a few months later, when he fell down in a fit in the street, and was picked up and taken into St. George's Hospital, where he died. There was, unfortunately, no *post-mortem* examination, so that the diagnosis is not quite certain, but I thought it a case of granular kidney. It is interesting to add that a brother of his died about the same time with kidney symptoms; and one of his sisters also was brought to me with persistent albuminuria.

Fits (Epileptiform Convulsions).—Fits are, of course, the common form in which acute uræmia manifests itself, but epileptiform convulsions are not rare as one of the early symptoms of the late

stage, independent of what is commonly understood by uræmic fits. This is an important point, for I do not think it is generally recognised.

The fits occur in groups, two or three a day possibly, and last for a day or two; they may then disappear, and not recur for weeks or months, when a second bout of fits occurs with as little manifest cause as before, and so on. Before long grave symptoms of the usual kind develop, and the case passes into its final stages.

Fits of this kind are, however, not peculiar to granular kidney, but may occur in chronic parenchymatous nephritis.

The attacks sometimes do not pass beyond the stage of unconsciousness, and may not be accompanied by convulsive movements.

In a case recently seen, what the patient described as fits proved to be curious transient attacks of right hemiplegia with aphasia, the pathology of which could not be determined.

Cerebral Irritation.—In connection with fits may be placed the curious attacks of cerebral irritation, which are not at all uncommon. They may take the form of outbursts of general nervous irritability, of emotional excitement, or of almost maniacal delirium. They may last a few days only or be prolonged for some weeks, and in this condition patients have been certified as lunatics. These attacks, like the fits, are at first separated by long intervals but gradually become more and more frequent, and finally end in a condition of chronic mental perturbation of either the excited or melancholic type.

In this connection it is interesting to recall the great frequency with which granular kidney is found in patients who die in lunatic asylums.

Acute Renal Toxæmia.—Acute Uræmia.

Of acute renal toxæmia—that is, of uræmia as we commonly understand the term—much need not be said, for it is with uræmia that so many cases of granular kidney suddenly end. The onset of the uræmic attacks may be the first grave symptom of the disease from which the patient is suffering. Thus it is not at all uncommon in hospitals to find patients brought in unconscious, and dying without the diagnosis being certain till after the autopsy, as between uræmia, epilepsy, or cerebral hæmorrhage, so suddenly may the attack come on and end, and

so little clue may the patient's symptoms give as to the real cause and nature of the disease.

The symptoms of acute uræmia have by no means that definite and uniform character which seems to be often assumed. The cases really vary very much from one another, almost as much as do cases of so-called diabetic coma.

Fits and coma are the two most characteristic symptoms, and yet patients may not have fits, or at any rate no marked convulsions, and they need not be comatose. A patient may be comatose without any fits, or at most with but very slight twitching, and the condition may then closely resemble apoplexy. In other cases the condition almost resembles that of narcotic poisoning. In others again, symptoms of the most profound collapse develop; the patient seems to have been suddenly poisoned, and presents symptoms very much like those met with in acute ptomaine poisoning.

Frequently signs of cerebral irritation develop, the patient becomes extremely restless, sleepless, and more or less delirious, and sometimes passes into a condition of noisy, active delirium not unlike delirium tremens, with which I have seen the condition confused. At other times the patient becomes violently maniacal, and is for the time a raging lunatic. If the patient do not die during this stage, the attack may subside, and in a day or two pass off. But even then the general condition rapidly deteriorates, and other symptoms develop which before long end in death.

The most interesting fact about uræmia in any of its forms in the course of granular kidney is that it may develop so suddenly and with little or no warning, in the midst of apparent health.

Whatever the form acute uræmia may take, the prognosis is as grave as it can be. If any one of the forms has a less grave significance than the others, it is perhaps that in which there are epileptiform convulsions, for uræmic fits may end in recovery now and then in granular kidney, as they more frequently do in acute nephritis.

In this connection the following case is of very great interest, for the attack was like one of apoplexy, though it proved ultimately to be uræmia. What is still more remarkable is that this patient recovered completely, and has been at his usual work and in his usual health up to the present time, now many months after the attack. Such a result, it is hardly necessary to state, is extremely rare:—

Samuel C., aged 43, a well-built and fairly healthy-looking man, was in good health and able to follow his usual occupation until May 1st. He then complained of feeling unwell, but of nothing definite; asked for food, but could not eat it; was extremely irritable, and his arms trembled a little. The next day he became somewhat rambling in talk, and by the evening was unconscious. He was brought to the hospital in this condition, and admitted as a case of apoplexy. The patient was quite unconscious, the face somewhat blue, the breathing stertorous, and the cheeks puffed out on expiration. The eyes and head had a tendency to turn to the right, but the legs and arms were both moved from time to time, so that the patient was not paralysed. The plantar and tendon reflexes were present on both sides. The arteries were thickened, tension high, pulse 120; there were slight traces of albumen in the urine, specific gravity 1020; the left ventricle was a little hypertrophied, the apex being in the nipple line. With these exceptions there was nothing to be discovered. The patient was treated in the usual way. In the evening it was thought the patient might be relieved by venesection, and accordingly about 20 ounces of blood were removed from the left arm; 5 grains of calomel were also given as a purge.

The temperature on admission was 100·6°, falling that evening to below normal. During the night the patient was very restless and continually groaning, and in the early morning of May 4th he became somewhat quieter, but was still unconscious, and shortly afterwards had five fits, each lasting about three minutes, and confined to the left side. The temperature during the night rose to 104·2°, but fell again in the course of the day to normal.

On May 5th the temperature, which had risen in the latter part of the preceding evening to 102°, was found to be normal, and from this time did not rise again. Breathing was quieter, and the patient now began to speak a little, and recognised his wife. He passed a fair amount of urine, which contained, as before, a small amount of albumen.

On the 6th he was better until the evening, when he became somewhat wandering and was very restless.

From this time he began gradually to improve, though for the first few days the improvement was slow.

On May 11th the patient, who had previously been taking hydrate of chloral and bromide, was now put upon nitrate of pilocarpin, one-sixth of a grain three times a day. From this time he rapidly improved, and on the 23rd was discharged well. He has been seen several times since, and continues well, and, though obviously the victim of granular kidney, has now no symptoms to trouble him.

This case shows that the acute uræmia of granular kidney may be recovered from, just as that of acute nephritis; but it is hardly necessary to add that recovery is much less frequent, for in most cases of granular kidney uræmia leads to death in a few hours, or at the most a few days.

Prognosis.

What with uræmia and other acute symptoms to which I have referred, I think we may say of advanced granular kidney as we can of diabetes mellitus—that the patient's life is of very uncertain

tenure, so sudden may be the onset of grave symptoms which end in death.

Yet, on the other hand, we know that in the absence of acute uræmia or serious hæmorrhage life may last for many years, especially if the disease be recognised early, and proper care taken in the management of the health.

Treatment.

If we knew the causes of the disease we might take steps to prevent it; but not only are the causes unknown, but the disease is not even recognised in most cases until it is already far advanced. Little or nothing definite, therefore, can be done at present in the way of *prophylaxis* or prevention. But as it is in the early stages that we are likely to be able to do most good, the importance of an early diagnosis is obvious.

When the disease is far advanced it cannot be cured, for scar tissue cannot be removed from the kidney by therapeutic means any more than from the skin or from the liver.

But because we do not know at present how to prevent the disease developing and cannot restore the diseased organs to their normal condition, it does not follow that treatment is of no avail. If we cannot treat the disease we can at any rate treat the patient; if we cannot cure the lesion we can at any rate attack the symptoms. If we can relieve the patient of the symptoms which trouble him, he will care but little for the morbid lesion; if we can cure his dis-ease, *i.e.*, discomfort, he will not much mind the disease.

We cannot cure myxœdema, but we now know how to relieve its symptoms. It is not too much, perhaps, to hope for similar success hereafter in the treatment of granular kidney.

The objects we should have in view in treatment are :—

(1) To prevent the disease becoming worse, and relieve the damaged organ in every way possible.

With this in view exposure must be avoided as well as fatigue of either mind or body; the general health should be kept at its highest level; the diet should be appropriate, and, where feasible, the winter should be spent in a warm, dry, and genial climate.

(2) To guard against the accidents specially likely to occur. These are failure of the heart and rupture of vessels.

With this object violent exertion must be avoided, as well as all excessive mental work and anxiety; the patient should lead an easy life, both physical and mental; and, where unavoidable illness arises, such as a severe accident or acute pneumonia, if the weak spot be remembered much may be done to diminish the risks.

(3) To counteract or relieve symptoms as they arise.

I have already drawn attention to the extreme variability of these symptoms, and the treatment must therefore be correspondingly various also; but it is often surprising when the cause upon which these symptoms depend has been recognised, that is to say, when granular kidney has been correctly diagnosed, how much may be done to give relief.

Of single drugs, I do not know of any one more useful than nitrate of pilocarpin given in small doses two or three times a day by the mouth, or in urgent cases *sub cutem*. Thus the headache, irritability by day and restlessness by night, vomiting and digestive disturbance, a foul tongue and a dry skin, may all rapidly yield to a dose or two of pilocarpin, and the patient be restored to comfort, or even threatening symptoms of uræmia be removed. There is one noteworthy fact about pilocarpin in chronic renal disease—namely, that it often does not produce the sweating which under normal circumstances and in similar doses it causes, and yet without the sweating its effect is striking.

I do not think it necessary to enter into details as to the ways in which these general principles of treatment can best be carried into effect, for on these points much will depend upon the individual case as well as on experience and good judgment.

There is, however, one line of treatment to which I wish more particularly to refer, for it has not so far been investigated as fully as it deserves, viz., the treatment of chronic renal disease by means of *renal extracts*.

I have already said that there is no positive evidence at present of the existence of an internal secretion in the kidney; yet the cachexia which develops in chronic renal disease bears a striking resemblance to that which is met with in Addison's disease or even myxœdema. Granular kidney also presents other resemblances with these diseases, in that the grave symptoms do not arise until the organ is very considerably diseased; in fact, as Bradford has

shown, it is not until about three-fourths of the total kidney substance has been destroyed that life becomes impossible.

In the case of myxœdema, there was the same absence of conclusive evidence until the administration of thyroid extract proved by its clinical effects that the symptoms depended upon the wasting of the gland. There can be no doubt clinically that the late symptoms of granular kidney do depend upon the disappearance of the glands, and it is therefore not at all improbable that the kidney has an internal secretion, though absolute proof is not yet forthcoming.

Renal extracts have not been used except in quite the latest stages of granular kidney at a time when probably the disease is too far advanced to be amenable to treatment at all. The subject is, of course, surrounded with difficulties; and yet, if we are to do any good at all by this or any other method, we must attack the disease before it has reached its later stages, and before the loss of nutrition and general cachexia are pronounced.

The first question is whether renal extracts administered by the mouth have any effect at all. I have already said that if nitrate of pilocarpin be given by the mouth or *sub cutem* to patients with granular kidney, the dose which under ordinary circumstances would produce free sweating often fails to produce this effect. The following experience is one which I have had on several occasions:—

A patient with granular kidney who had been taking nitrate of pilocarpin regularly was given renal tabloids two or three times a day. The dose of pilocarpin had hitherto caused no sweating; but within 24 hours or so after taking the renal tabloids sweating became marked, and followed each dose to such an extent that on some occasions it had to be stopped. As soon as this was done the sweating ceased. After a short interval the pilocarpin was repeated, and the sweating returned. The pilocarpin was then continued and the renal tabloids stopped, and the sweating again ceased. The administration of renal tabloids, therefore, did in these cases modify nutrition in some way so as to permit the nitrate of pilocarpin to have its normal action.

The most striking instance of the effect of the renal tabloids occurred in the following case:—

Ellen B., aged 13, was under treatment for what appeared to be acute nephritis. After some weeks' illness she began to develop uræmic

symptoms ; the amount of albumen increased considerably ; the amount of urea fell to 0·8 per cent. ; there was a good deal of vomiting and abdominal pain, some diarrhœa, and also some bronchitis. A little later headache set in, vomiting became more troublesome, and she began to have fits. Under treatment the fits subsided, and in the course of a week or so she was in much the same condition as before the fits commenced. As she seemed to be going downhill, I administered renal tabloids, giving her 5 grains twice a day. Coincident with this she began to improve ; the quantity of urine a few days after the first administration increased and reached 47 ounces—the largest amount she had ever passed while under treatment, the usual average hitherto having been not more than 15 or 20 ounces. With the increase in the urine the amount of albumen greatly diminished and the œdema began to disappear. In the course of a month the urine averaged 40 ounces in amount, 1012 specific gravity, and not more than 0·5 per cent. of albumen. Soon after she was well enough to leave the hospital. Some months later she returned again in a condition of dropsy and chronic uræmia. She again took renal tabloids with great benefit, and again left the hospital, except for the albuminuria, well. A third time she was brought to the hospital, this time in uræmic convulsions, and died in a few hours.

I have had two or three other cases of the same kind, and in all the results have been the same, namely, an increase in the amount of urine and an improvement in the general condition. This improvement followed, and appeared to be due to the action of the remedy.

The use of renal extracts is still a purely experimental stage. Judging by the analogy of myxœdema, it would be in the cases of chronic renal cachexia only that we should look for striking results, and this requires early and correct diagnosis. In acute uræmia there is so little time to act that recourse must be had to other and more active measures.

I have now finished the task set me. My choice of granular kidney as the subject was inspired by the hope that what had interested me would prove of interest to others also. I trust I have not misjudged. I have not hesitated to express my own opinions, even at the risk of appearing egotistic, though, I trust, with sufficient modesty to escape the charge of dogmatism. On a subject so full of difficult and contentious questions, I cannot expect that my views will commend themselves to all. Yet defined opinions challenge criticism, and criticism tends to advance knowledge.

February 27th, 1899.

AUTO-INTOXICATION AND ITS RELATION TO THE TREATMENT OF DISEASE.

By Sidney Martin, M.D., F.R.S., F.R.C.P.

By *infection* is meant the invasion of the body by a living germ, which, by itself or its products of activity, injuriously affect the tissues. By *intoxication* is meant the poisoning of the body by chemical agents, usually the products of activity of the living germ.

No definition of either of these processes can, however, be rigid. A typical example of infection is seen in anthrax, where, from the primary infection, the bacillus invades the body. In other cases, however, an infective agent may invade a tissue, and in that tissue manufacture its products, which poison the body; thus intoxication is added to infection. A good example of this occurs in tetanus, where the bacillus is localised to the seat of injury, and the toxic substances are absorbed from this spot. The invasion of the body by pus cocci may result either in a general pus infection, a pyæmia, or a localised infection, from which intoxication results.

These examples are quoted as bearing on the question of *auto-intoxication*, or self-poisoning. In this case the body manufactures its own poisons, which result in an affection of the tissues; they act as protoplasmic poisons. Many different conditions are included under the heading auto-intoxication—conditions which are in some instances so widely different, as we shall see, that it is very doubtful whether the term ought to be retained as applying to all of them, either from a pathological or a clinical point of view.

In order to discuss the subject, however, it may be grouped under three chief headings:—

1. Auto-intoxication of gastro-intestinal origin.
2. Auto-intoxication occurring in the course of chronic disease, such as chronic renal disease, diabetes, and degenerative lesions of the brain.

3. Auto-intoxication in association with the glands of the body —the thyroid, suprarenal, and pituitary body among the ductless glands, and the liver, pancreas, and testicles among the glands with ducts.

It is necessary to premise that the activity of a tissue in disease may be altered (and so produce symptoms) in various ways; *e.g.*, by interference in one way or another with the vascular and nervous supply, by the accumulation in it of the products of its own activity, by the preponderance in it of certain mineral salts, the chief of which are those of potash, and by the action on it of some poison (bacterial or not) brought to it by the blood stream and originating either outside the body or within it. It is clear, therefore, that the determination of the cause of this effect on the tissue may be very difficult if not impossible. To illustrate this, take the muscular tissue. This may be rendered inert to stimulation by repeated shocks sent through the nerve; the resulting repeated contractions of the muscle lead to fatigue and the nerve-muscle no longer responds to stimulation until it has had time to recover. But in disease the nerve-muscle may be affected in the same way by poisons, or by alteration of the vascular supply. So that what we observe as a symptom may be produced by many different causes, which may throw an organ or tissue out of activity.

A poison, it must be remembered, does not necessarily kill; and death of the body—somatic death—although in some instances it is to be ascribed to the specific action of the poison on vital centres (as in the case of prussic acid or snake-venom acting on the centres in the medulla) is in many cases the final effect of the poison—the result of an action on various tissues and organs. This is particularly to be noticed with the poisons that act in disease. A toxic agent may excite, paralyse, or kill a tissue, but one of the most important effects to be considered from our point of view is the specific or selective action of the poisons in disease on tissues: some selecting the heart and circulatory system for their action, others the peripheral nerves and muscles, others again the glands, *e.g.*, the kidneys, and others the nerve centres. Such substances must be considered as poisons, for although they may not cause somatic death, their presence in excess in the body alters the physiological relations of the tissues and fluids of the body and leads to chronic disease.

1. Auto-Intoxication of Intestinal Origin.

The chief examples of auto-intoxication of intestinal origin are those to be ascribed to bacterial decomposition of the gastro-intestinal contents, as distinguished from bacterial infection of the intestine. By some (Bouchard, *e.g.*) even typhoid fever and cholera have been discussed as examples of auto-intoxication, but there is no more reason why these diseases should come under this heading than diphtheria, which is an infection of the mucous membrane higher up.

Bacteria are taken in with the food; some are killed during gastric digestion, with others the growth is simply inhibited. As the food passes along the small gut the bacteria increase somewhat, chiefly the acid-forming varieties, but not to any great extent. The great increase occurs in the large intestine, and results in the formation to a greater or less extent of putrefactive products. With the exception of the bacillus coli communis, most of the bacteria in the intestinal contents are not to any great extent pathogenic, as long as they remain in the intestine. In cases, however, where the lumen of the gastro-intestinal tract is obstructed in one or other way, putrefaction of the intestinal contents occurs, and the products being absorbed poison the body. This is referred to as auto-intoxication—in some cases as sapræmia, in others as stercoræmia. The symptoms of poisoning may result simply from fæcal impaction, or from obstruction due to organic disease—strangulation, stricture, &c., the symptoms in the latter case being the more severe. Pathologically this process of intoxication does not differ from other cases of bacterial intoxication, and the process frequently passes from one of intoxication to one of infection of the intestinal wall or of the peritoneum. It is better, I think, to discard the term "auto-intoxication" for those bacterial intoxications that occur in the intestine, and to range them with the other bacterial processes which occur in disease, and this would be an advantage even from the clinical point of view.

Another form of gastro-intestinal auto-intoxication must be considered, viz., that resulting from the indigestion of food in the stomach or intestine. Besides the local symptoms which are observed in such conditions, there are others which are described as "remote" or "reflex," such as headache, malaise, irregularities

of vision, giddiness, and those referable to the nervous system generally. Some of these have been ascribed to auto-intoxication by means of the normal or abnormal products of digestion. The question is an extremely complex one, inasmuch as most of the symptoms are explicable as reflex nerve effects from the irritated organs, effects which may result in disturbances of the circulatory system or of the central nervous system. It is, moreover, to be remembered that indigestion of food means frequently either a deficiency of a proper amount of nutritive material, or the absorption of too large a proportion of one or other food stuffs, whether proteid, fat, or carbohydrate. In either case there would be a distinct effect on the tissues of the body which would suffer in nutrition, and so exhibit the symptoms of disease. While this subject, therefore, is very complex, it must be discussed in reference to auto-intoxication, since during the normal processes of digestion, bodies are formed which have poisonous properties. Some years ago, Brieger stated that he had obtained as a product of artificial peptic digestion an alkaloidal body, peptotoxin, which was poisonous. It was suggested that this body was formed by pepsin under certain conditions, and its occurrence was said by some to explain the symptoms of indigestion. Salkowski, however, showed that in peptic digestion, when care was taken to exclude bacterial decomposition, no alkaloidal body was formed. The poisonous products which are found in both peptic and pancreatic digestion are the albumoses and peptones, which when injected into the circulatory system of certain animals produce a great lowering of blood-pressure, cause fever, and prevent coagulation of the blood. In functional disorder of digestion these albumoses are sometimes absorbed as such; being found in small quantities in the urine in certain cases of dilatation of the stomach, and other cases where absorption appears to be disorganised. Normally in the process of absorption these albumoses are transformed by the mucous membrane of the stomach and gut into the proteids of the body, and are not found in any of the tissues or liquids of the healthy organism. To what extent or with what regularity they are present in the blood in these cases of disordered digestion it is impossible to say, as definite data are wanting, but it is quite possible that their presence in part accounts for some of the symptoms of disordered nutrition observed. In not a few cases, however, in which they

are found in the urine, pronounced symptoms which might be ascribed to their presence are wanting; this is a fact I have noticed several times.

2. Auto-Intoxication in Chronic Disease.

We have seen that the chief examples of so-called auto-intoxication from the gastro-intestinal tract are cases of bacterial intoxication, and that the term in this case is somewhat a misnomer. Toxic symptoms, however, occur in the course of disease in which the poisons do not apparently come from without, nor do they originate in the gastro-intestinal tract. Some of these cases must probably be ascribed to an auto-intoxication in which the poisons originate in the tissues of the body, and are not due to bacterial activity. The question arises, Does the healthy body contain any poisonous substances? The proteid substances present in the blood and organs of mammals are non-toxic: this being more particularly the case with the albumins and the globulins. In lower animals these proteids may be toxic, *e.g.*, the blood of the conger-eel and muræna and the venom of snakes. The class of nucleo-albumins, however, which are obtained from lymph glands chiefly, and have been worked at chiefly by Wooldridge and by Halliburton, have a distinct physiological action chiefly in causing coagulation of the blood when injected into the circulatory system. The nitrogen extractives of the body—urea, uric acid, creatin, creatinin, sarcosin, &c.—which are the products of normal proteid metabolism, are but slightly toxic; potash salts are poisonous, acting as cardiac depressants.

In disease, it is quite reasonable to suppose that the abnormal preponderance in a tissue either of the products of proteid metabolism or of salts of potassium may have an effect on the activity of the tissue or organ, and cause a serious functional disturbance of nutrition. Such a condition would include the abnormal accumulation of urates in the body, as well as their change of chemical constitution, causing them to be deposited in the tissues, as in gout.

Another important aspect of the question has, however, been of late years brought prominently forward, viz., the formation of poisonous products from proteid substances. By the action of bacteria (either directly or by means of ferments they secrete)

proteid substances split up, forming two classes of poisonous bodies, one belonging to the class of digestive products (albumoses), the other non-proteid, and corresponding to the class of the so-called nitrogenous extractives of the body. The question arises, Can the proteid or other substances in the body in chronic diseases which present symptoms of intoxication split up into poisonous substances without the aid of bacteria? This question is at present unanswered; no such poisons have been separated.

There are two chronic diseases (which may also appear in an acute form) in which are observed symptoms apparently due to intoxication; these are chronic disease of the kidneys (Bright's disease) and diabetes.

In chronic Bright's disease the evidences of intoxication or poisoning are seen chiefly in the phenomena of uræmia, in which convulsions, coma, and disordered respiration, such as Cheyne-Stokes breathing and paroxysmal dyspnœa (renal asthma), may occur without there being any gross or microscopic lesion to account for the symptoms. Uræmia is a part of Bright's disease, and is to be distinguished from the direct effects of high arterial pressure and diseased vessels, *i.e.*, hæmorrhage. By some the phenomena of uræmia are considered as the result of high arterial pressure, but this appears to be no solution of the question, and the symptoms, indeed, are more naturally to be ascribed to the selective action of a poison or poisons on the cerebral centres. Albuminuric retinitis and optic neuritis, which are observed in Bright's disease, may also be the results of intoxication, although in this case again high arterial tension has been supposed to play the main part. Peripheral neuritis is sometimes observed in the course of chronic Bright's disease; in some cases chronic alcoholism is the most obvious cause, but in one case examined by me, in which there was extensive degeneration of the nerves, the immediate cause of death being respiratory paralysis due to the affection of the intercostal nerves, alcoholism could reasonably be excluded as a cause of the neuritis, and it appeared probable that this was a part of the phenomena of the renal disease.

I do not quite follow Bouchard in his experiments on the toxicity of urine. He has determined by physiological experiments the presence of several different poisons in the urine. It is too large a question to enter into fully here, but one of Bouchard's points is that there is evidence of uræmia being a

condition of intoxication, in the fact that during its presence the normal toxicity of urine is greatly diminished, showing the retention of poison in the body. In the absence, however, of any demonstration and isolation of poisonous substances in the urine of cases of chronic Bright's disease, the physiological effects of which resemble the condition of uræmia, the conclusion cannot be considered as very definite.

In diabetes, the evidences of intoxication are chiefly the coma which is observed. Although there are evidences of the formation in the body of substances not present in health (acetone and oxybutyric acid), these have not a pronounced toxic action, and no poison has been isolated which can be said to account for the phenomena. Another evidence of intoxication in diabetes may be said to be the neuritis which sometimes occurs in cases in which chronic alcoholism or a previous infective disease can be excluded. Peripheral neuritis, a parenchymatous degeneration of the nerves, is the result, in the majority of cases, of toxic action, as witness its occurrence in alcoholism, in lead and arsenical poisoning, and in diphtheria. It may result from the action over months or years of small quantities of poison, and it is probable, therefore, that the amount of the poison present in the tissues at death may be so small as to be almost impossible to isolate. Similarly with the coma; this may be the final result of the action of small doses of poison.

In atrophy of the brain, Dr. Mott has found that the cerebro-spinal fluid has a distinct physiological effect mainly in the lowering of blood pressure: the general action being suggestive of that of cholin, the source of which would be the lecithin. The importance of this line of research cannot be over-estimated: in chronic degenerative disease of the brain, toxic phenomena, in the form of convulsions, &c., may occur without a gross lesion being present to account for them. In many other conditions occurring in chronic disease, auto-intoxication may be found to play a part. Accurate information is, however, at present wanting.

3. Removal of Normal Glands Affecting Nutrition.

It is probable that in the diseases discussed we have two examples of auto-intoxication. Another aspect of the subject must now be considered, viz., the effect on the body of the removal

of normal glands. The results obtained have given quite a new aspect to some diseases, and have done something to explain some of the obscure forms of nutritional disease which are frequently observed. The subject is too large to discuss in anything like detail, but two aspects of it may be noted as important from our present discussion.

1. The removal of these glands from a normal animal results either in death or in a profound change in nutrition.

The removal of the pancreas, the pituitary body, the suprarenal bodies is incompatible with life. The complete removal of the thyroid results in the well-known phenomena exhibited in myxœdema, as shown in grave defects in nutrition. The removal of a certain proportion of the kidney substance also results in remarkable chemical changes in the body.

The symptoms observed after removal of the pancreas are glycosuria, polyuria, and wasting; after removal of the pituitary body there is a lowering of the body temperature, anorexia, and lassitude, muscular twitchings, nervous spasms, and dyspnœa, symptoms which are frequently produced by intoxication; after removal of the suprarenal bodies there is extreme muscular weakness, loss of vascular tone, and anorexia. These examples serve to illustrate the profound effect the removal of those organs have on the body, and their bearing on disease.

2. The effect of their removal has been ascribed to the absence of an internal secretion which is normally given to the body, and which is essential for the carrying out of the usual physiological processes.

On the other hand, the phenomena following removal have been ascribed to auto-intoxication; these glands remove some poison which is inimical to life.

It is important to remember that the extracts of certain of the glands have been proved to have a distinct physiological action of their own. Thus thyroid extract in the healthy individual not only causes a fall of blood pressure, but profoundly affects nutrition, causing wasting; extract of pituitary body causes a well-marked rise of blood pressure; extract of the medulla of the suprarenal bodies causes a similar and remarkable rise, with a distinct effect both on the heart and the blood vessels, and affects the contraction of muscular tissue like the alkaloid veratrine, viz., in greatly prolonging the phase of contraction.

Inasmuch as these normal glands possess these substances with such a well-marked physiological action, and that the removal of these glands is either fatal or produces a diseased condition, it is fair to suppose that in the normal body these glands do form substances (internal secretions) which are essential in maintaining the complex physiological processes of health. But it must be remembered that a substance with a physiological action may, in excess, become a poison, and that these internal secretions may be actually the cause of disease, not only by their amount being diminished, but also by its being increased. Indeed, in exophthalmic goître we have a condition which is in many ways a marked contrast to myxœdema, and it has been suggested that whereas myxœdema is an example of diminished, exophthalmic goître is an example of increased activity on the part of the thyroid gland.

The treatment of disease, if it is ever to be rational, must be based on accurate knowledge of diseased conditions. Disease does not always show itself as a tumour or an abscess: there are many conditions which are still very obscure, and on which but little light has been thrown by a mere study of symptoms, important though this is. The history of myxœdema, as well as of the investigation of the physiological *rôle* of the suprarenal bodies, shows that a detailed study of the obscure conditions which we have discussed may result in the future in important discoveries in their treatment.

The PRESIDENT congratulated the author on the able and convincing way in which he had brought forward his communication, and he asked whether rickets was not probably a form of auto-intoxication from the gastro-intestinal tract. In the routine treatment of rickets one began with a dose of rhubarb and soda to clear out the alimentary canal, and this generally produced an extremely offensive evacuation, after which the child usually began to improve. He remarked that the author had alluded to peripheral neuritis as occurring after diphtheria and other conditions, but he had not expressly referred to the neuritis that occurred after influenza, which must be due to some form of intoxication. In the signs of fatigue and headache experienced after unwonted athletic exertion he thought he saw signs of auto-intoxication.

Dr. MOTT said that when he was appointed pathologist to the London County Asylums it occurred to him that the lines upon which they ought to work were bio-chemical rather than histological. The difficulties of such a research were of course very great. He at one time undertook researches upon metabolism, and had had to give them up as it was found impossible to obtain all the excreta. The first thing that was advisable was to take a disease in which there were well-marked organic changes,

so he took general paralysis, and as the patients invariably died he was enabled to examine the tissues afterwards. The degeneration of nervous tissue led to the formation of a fatty substance which stained black with osmic acid after hardening in Muller's fluid. Ordinary myelin did not stain in this way, so that there had evidently been some decomposition probably into glycero-phosphates and cholin. The former were probably converted into phosphates and passed away in the urine while the cholin would be absorbed and circulated in the blood. The next thing he did was to inject normal cerebro-spinal fluid, but it gave no result whatever. In conjunction with Professor Halliburton he had for the past three years undertaken an experimental research upon the effects of intra-venous injection of cholin and neurin. They invariably found, however, that the cerebro-spinal fluid of people who died of general paralysis brought about a fall in the blood pressure. Cholin had the same effect. They, therefore, concluded that there was probably cholin in the fluid. This assumption was proved to be correct by their having been able to crystallise out the platinum salt of cholin, and this proved that the phosphoretted fat had broken up into a simple neutral fat and cholin. One could see this fat, which stained black, in the perivascular spaces and in the tissues generally. They also found a small quantity of nucleo-proteid which, as the author stated, was a powerful coagulating agent, and he suggested that possibly some of the complications of general paralysis might be due to this body causing venous stasis, because one of the most noticeable conditions microscopically was the distension of the veins. The venous stasis amounting sometimes to thrombosis would produce corresponding local cerebral anæmia. They had thoroughly worked out the action of cholin, but whether it had the importance they attributed to it he could not say. It caused dilation of the splanchnic area. There was no doubt that this substance was a poison, not very powerful perhaps, but if it accumulated in the blood it might act in a poisonous manner, so that it was a probable source of auto-intoxication. Neurin, which was readily converted into cholin, was a powerful poison, producing first a fall and then a rise of blood pressure, accompanied by respiratory spasms. Neurin and cholin belong to the trimethylamine series, which includes muscarin, and there are two other bodies of the series which are quite innocuous. They had also injected small doses of glycero-phosphoric acid into the circulation without result. The two substances which collected in the cerebro-spinal fluid which were poisons were cholin and nucleo-proteid. At the same time he was working to ascertain the cause of death in general paralysis. If one only looked at the brain one might have a difficulty in saying what such patients actually died from. In 150 cases he had made careful observations, and in a large proportion he had found fatty degeneration of the heart-striped muscles, very acute in some cases. He had been rather led astray by this to begin with thinking it was caused by cholin. He took the blood pressure in a number of cases during the epileptiform seizures and instead of a fall in the blood pressure he found a rise, an extreme rise. He used Barnard and Hill's sphygmometer, which sometimes indicated a rise to 200, the normal being 120, and even in cases where the heart sound was so weak as hardly to be audible the blood pressure was frequently above normal. It might be said that the fatty degeneration of the muscles and of the heart was due to changes in the nerves, but he had examined a large number of vagi, phrenic, and other nerves without finding any evidence of degeneration to account for the condition of the muscles. During

these observations he came across five cases of status epilepticus in healthy men previously working in the wards or in the fields. One was 18 years of age, and two days before he died he was seized with fits which continued until death. His temperature went up to 105°. He examined the muscles, liver, kidneys, and heart, and found intense fatty degeneration. 1 in 5 of the fibres of the diaphragm and 1 in 10 in the other muscles had undergone degeneration, the liver was almost quite black with the stain, and there was considerable change in the kidneys. This was noted in all five cases. Evidently some process of auto-intoxication had taken place and a vicious circle been established. It might mean imperfect metabolism but that also amounted to auto-intoxication. He referred to the experiments of Araki, which showed that after continuous epileptiform seizures sarcolactic acid was found in the urine which one also got in phosphorus poisoning. Although he had not been able to inject blood and saline solutions of organic extracts into animals the line he thought was one likely to prove fruitful. Another point was that in general paralysis there was often fever even before the convulsions began (100° to 101°). Another factor with regard to auto-intoxication was that the injection of a large quantity of saline solution, by way of enema or otherwise, would often prevent or check the fits. This was in fact the treatment which he would recommend under all such conditions.

Dr. Washbourn suggested that the term auto-intoxication was very misleading for it was used to express two distinct conditions—the one the absorption of substances, either living or dead, from the intestine, and the other certain changes which occurred in chronic diseases of various organs, such as the liver and kidney. Absorption from the intestine in many cases was really not absorption of poisonous products from the intestinal canal but an infection by bacteria, and with regard to cholæmia and uræmia it still remained to be proved that these conditions were due to the circulation of poisonous substances in the blood. The first group was very interesting for many reasons. In the intestines there were a large number of bacteria which were known to be capable of setting up grave symptoms if they got into the tissues of the body, and there was good evidence that these bacteria could produce poisonous substances during their stay in the intestine which, if absorbed, produced injurious effects. Therefore the body must possess a powerful means of defence against the invasion of bacteria from the intestine and also against absorption of their products. As to the mode of protection against invasion of bacteria from the intestine, he thought that much still remained to be learned. It could not be that the bacteria actually invaded the tissues of the intestinal walls and were destroyed by the cells and fluids of the body, as occurred when they were introduced under the skin. It was probably due to some chemiolactic influence which prevented them passing through the mucous membrane. He asked how it was that the intestine, while absorbing the products of digestion, did not absorb the poisonous bacterial product. The albumoses and peptones were absorbed, but these were not. There must consequently be some defence against these substances, of the nature of which we were still ignorant. Then as to the pathogenic bacteria frequently found in the intestinal tract without producing any effect, the diphtheria bacillus for instance. Was it that the bacilli produced the toxins so slowly as to give rise to immunity, or was it that the body possessed some power by which it could inhibit the production of toxins? An alteration in the nutrition

of the intestine at once annulled the protective mechanism, so that not only bacteria but also toxins passed through the intestinal wall. With reference to the various forms of auto-intoxication which occurred in disease of important glands, he thought it was very necessary to remember that almost all the glands of the body had at least two functions, the one that of excretion or secretion, and the other a function connected with the metabolism of the body. In cholæmia and uræmia the explanation probably was, not that certain substances failed to be excreted, and therefore accumulated in the tissues, but that there was an alteration in the metabolic functions of the organs occasioning profound changes in nutrition. In uræmia, for instance, the principal feature was not an accumulation of urea in the blood, but alterations in the metabolism of the body due to injury of the kidney cells. Both cholæmia and uræmia, in fact, were primarily due, not to intoxication but to alterations in nutrition. Such an alteration of nutrition might at a late period give rise to the formation of poisonous products by other tissues than the kidneys, and then an intoxication might ultimately ensue.

Dr. Sidney Martin, in reply, agreed that it was a mistake to include under the head of auto-intoxication processes of bacterial origin. He had not discussed rickets, for he knew very little about it. He had not mentioned influenza neuritis, but what he wished to point out was that peripheral neuritis was a sign of poisoning in the case of influenza, it was the result of bacterial intoxication. Dr. Mott's work was interesting in that it showed the existence of poisoning in the course of a chronic disease. Of course bacterial products of digestion were sometimes absorbed, as evidenced by the presence of ethereal hydrogen sulphates in urine.

March 13*th*, 1899.

THE OPERATIVE CURE FOR INGUINAL HERNIA, AND AFTERWARDS.

By John Langton, F.R.C.S.

Shortly after the election of your President to his high office he requested me to introduce a discussion "On the operative cure for inguinal hernia, and afterwards." Feeling that our President's request was equivalent to a command I acquiesced with some misgiving which will be apparent in alluding to the permanent results of the operation. In talking over the scope of the discussion we both felt, at any rate I did, somewhat strongly, that it should be confined to the consideration of the operative cure for inguinal hernia, as the wider field of the operation for femoral and umbilical hernia would probably lessen the value of the discussion.

I take it that it was the intention when this subject was proposed that the discussion should be the outcome of our practical experience of the operation and not an academic or abstract consideration of its literature. It is not surprising in view of the wide distribution of hernia, amounting to nearly 6 per cent. of the entire population, of its far-reaching effects and of its constant danger to life from strangulation, that its treatment should have for many centuries engrossed the attention of surgeons and I regret also of quacks, and various have been the operations advocated for the cure of the disease.

Nearly 2,000 years ago, Celsus, all of whose books save one have been lost, very fully and accurately described this lesion, and for its cure he advised the separation of the sac from its connections, its ligature, the removal of the testis and some portion of the scrotum, in order to secure a dense scar over the external abdominal ring, and thus prevent any further extrusion. Later on Albucasis, following the advice given by Celsus, advocated the use of the actual cautery and the searing of the tissues of the cord as high up as possible. Shewing how history repeats itself, we find this practice again brought into fashion as late as the year 1726 by Houston, who strongly urged the revival of the practice of the " bold and wise ancients."

The formation of a dense scar over the abdominal ring seems to have been for many centuries the sole object of operating surgeons and the firmer the scar the greater the prospect of cure, for during the reign of George I a physician—Little by name—secured a handsome reward for his nostrum, which consisted in repeated applications of oil of vitriol, thereby producing a dense scar. He is reputed to have received from a grateful Sovereign the sum of £5,000 and an annuity of £500 for his lifetime.

Not advancing much above the practice of Celsus, we come upon a new era in the treatment of hernia, in the ligation and excision of the sac, a method I assume adopted by the Albanians when they operated on the secretary of Demetrius Cantimir, a Moldavian prince, a very amusing description of which may be found in his ' History of the Ottoman Empire,' where the method is described as " coarse but successful."

Surgeons even in comparatively modern times did not view with favour any operation for the cure of hernia. Cooper and

Lawrence, both largely ahead of their day, dreaded any interference with the sac, and owing to the mortality and the failure of the proceeding the operation fell into discredit. Nor indeed could the result have been otherwise, for relapse of the hernia must necessarily occur if the aperture through which the extrusion first emerged was not securely closed. Lawrence, with his characteristic surgical sagacity, duly recognised this shortcoming, but he does not seem to have devised or recommended any advance over the old operation of ligation and excision of the sac. He says, as if anticipating a not unimportant part of the present operation: "But in truth something more is required, we want a remedy that should contract the tendinous opening" —he means, I take it, the external abdominal ring—"for while that remains preternaturally large a new protrusion is a highly probable occurrence."

It appears to me that up to 1835 no attempt had been seriously made to occlude the inguinal canal. In this year Gerdy described his operation for invaginating an inflamed piece of scrotum into the ring and canal which was then retained by sutures. His method was improved by Wützer, who invaginated the scrotal tissues into the canal and maintained them in this position by an instrument through which a needle was passed. His success, however, was not great, and his operation was much improved by Mr. John Wood, who described in 1857 his now well-known operation. Mr. Wood must be regarded as the pioneer of a fresh advance in the scientific treatment for the cure of hernia, and he claims a high percentage of permanent cures amounting to nearly 70 per cent. of all cases operated on. This success does not appear to have been achieved by other surgeons in America, for Dr. Cheever, of Boston, stated in 1870 that the statistics showed barely 25 per cent. of cures, and he reports 20 cases of operation with two deaths.

The open method of operating has now been the practice for over a quarter of a century, since surgeons recognised the uncertainty and incompleteness of subcutaneous operations for the permanent cure.

Methods of the Operation.

It would be alike tedious and fruitless for me to review at length the various operations and their countless modifications advocated

by various operators. Each surgeon, as his experience ripens, sees weak points in every set operation which demand more or less important modifications during the course of the operation.

The following methods of procedure appear to me to involve primary and definite principles which may conveniently be arranged under the following heads :—

(*a*) The operation which aims at the reconstruction of the inguinal canal with transplantation of the vas deferens and its vessels to the upper angle of the incised aponeurosis, and so to form a new inner ring ; a method advocated by Bassini, Halstead, and others.

(*b*) The operation, as practised by Macewen and Ball, though differing from each other in many details, aims at utilising the whole or part of the hernial sac to form a pad or buffer within or close to the inner ring, so that the peritoneal fossa usually found here is converted by the sac-boss into a convex surface. This, when firm, effectually withstands the ever downward pressure of the abdominal viscera, and so prevents a relapse of the hernia.

(*c*) This method, which is comparatively simple, consists in ligature of the hernial sac close to the inner ring, its excision on the proximal side of the ligature, and the reconstruction of the inguinal canal.

(*d*) The plan advocated by Kocher and Treves consists in the partial removal of the sac, its ligation and torsion, followed by its withdrawal through the conjoined tendon and the inner pillar of the external ring and its retention in this position (Treves) or by passing the sac through the abdominal parietes and bringing it out through the aponeurosis of the external oblique internal to the anterior superior iliac spine (Kocher).

These methods have certain definite objects in common, the most important being the closure of the internal ring and the obliteration of the canal. In others, details in the technique are supplemented, such as the formation of a firm buffer at the inner ring by the hernial sac, the reconstruction of the canal, or the removal of the neck of the sac from its acquired position.

The set methods of operation have been, in many ways, materially improved by more or less important additions in dealing with the sac and the canal. In my judgment these numberless modifications are confessions of weakness as to the complete efficiency of the operative cure.

The ever-varying anatomical and pathological conditions met with during the operation render it impossible to determine in advance the exact method best adapted for each case, until the parts are exposed and their relations and conditions thoroughly defined.

The operation I recommend, and perform if possible, is the one advised by Bassini, with certain modifications, since it, in my experience, affords the best results. After exposing the canal by incising the aponeurosis of the external oblique in the usual manner up to the inner ring, I carefully dissect out the sac, which, if scrotal, is cut through close to the outer ring, and the scrotal portion tied and allowed to drop into the scrotum; but if small the hernial sac is left to be utilised later on. The sac is then carefully exposed up to its neck and separated from the margins of the inner ring, when it is twisted on its own axis as far as it will go. Retained in this condition of extreme torsion by a pair of Spencer Wells' pressure forceps it is transfixed by a Hagedorn needle threaded with a long piece of sterilised kangaroo tendon. The transfixed twisted sac is then ligatured in two halves, and then in its entirety. I then thread a full-curved Hagedorn needle with one end of the tendon suture, and pass it through the lowest border of the inner ring, through the drawn-down strong muscular fibres of the internal oblique and transversalis muscles, and then, curving the needle upwards and outwards, I bring it out through the aponeurosis of the external oblique half an inch above and to the inner side of the upper angle of the incised aponeurosis. The remaining thread is passed through these strata in the converse direction, and its end is eventually brought out through the aponeurosis half an inch to the outer side and above the upper angle of the incision. The sac on the operator's side of the ligature is then divided and passed through the inner ring as far as it can be passed, when the two threads are tied, but not too tightly. The position of the vas I do not alter, for wherever placed it must create a weak spot. Any division of the muscular fibres of the internal oblique and transversalis is to be deprecated, for all that is necessary to thoroughly expose the inner ring can be as well effected by drawing up the muscle by a small retractor. The value of these muscles in their support of the inner ring, especially during violent exercise, cannot be over-estimated. They are the natural supports of this weak spot, and they in great

measure prevent the extrusion of a hernia through the inner ring. If they are incised this area is unsupported, for a muscle weakened by division is rendered thereby less efficient than a sound one. It is rarely necessary to cut these muscles in order to expose the inner ring.

The canal is then sutured in the usual way with kangaroo tendon used as a continuous suture. I believe that the obliteration of the canal is more imaginary than real; it is narrowed no doubt, but not obliterated. Nor, indeed, could we expect any other result when we take into consideration the structures of which the posterior wall of the canal is composed.

I prefer to use the tendon suture as a continuous rather than an interrupted one, for the coaptation of the canal walls is more firm and complete, and it has, moreover, the advantage that the number of knots is reduced.

Lastly, I sew up with a continuous tendon suture the divided aponeurosis of the external oblique, passing my threads about a third of an inch beyond the edges, so that when the suture is tightened the aponeurosis becomes shortened and taut. The continuous sutures should not be too tightly drawn up, as this interferes with the proper nutrition of the sutured aponeurosis, and not unlikely ends in suppuration.

When dividing the different strata during the operation I take care to cut at different levels, otherwise the lines of incision will constitute the weakest part of the united abdominal strata.

Drainage tubes are rarely needed, but occasionally when the bleeding has been profuse and the hernia large a cavity is left which it is well to drain for the first 24 hours.

In young adults we not infrequently find, on opening the sac, a piece of sticky unaltered omentum lying at the neck of the sac, which is easily returned into the peritoneal cavity, but as promptly descends when pressure is removed. If this is left it easily acquires adhesions to the peritoneum close to the inner ring and may in future years be a source of danger in giving rise to internal strangulation. It is best, therefore, to drag down this omentum for a considerable distance, and when ligatured securely to return it into the peritoneal cavity.

I have come to the conclusion, after many years' experience, that Bassini's operation, modified in the way I have described, affords the best prospect for the patient's cure. In the continuous

tendon sutures we possess the most secure support, as it fulfils all the requisite requirements. With it there is less trouble from suppuration, and if this does occur, as occasionally it will, it is neither so persistent nor so intractable as with the silk suture.

The subsequent course of treatment must follow the ordinary surgical rules of abdominal operations. Rest in the recumbent position for a month is a very important element in the treatment, and though often irksome to the patient it is essential, so as to allow the plastic exudation to become firmly consolidated before locomotion is permitted.

Suppuration.

The ideal operation for the cure of a hernia is an aseptic one, and therefore a non-suppurative one. Occasionally, through some aseptic shortcoming, the incision fails to unite by primary intention in its entirety. The occurrence of this misfortune varies in its frequency and extent in surgical practice, and one cannot but be impressed with the widely divergent results recorded in the literature of the subject by various operators.

Dr. Bull, in the 'Transactions of the New York Medical Association, 1889,' states that in 69 cases operated on, primary union occurred in 32 cases — a percentage of 46·4; while suppuration took place in 37 instances—a percentage of 53·6. In the recorded experience of Dr. G. W. Jones, of Cambridge, U.S.A., we find that out of 37 consecutive operations suppuration took place in only one patient.

The results of our experience at St. Bartholomew's Hospital, taking only those in which special notes of this occurrence are recorded, shew that the non-suppurative operations amounted to 83·5 per cent. and the suppurative to 16·5 per cent.

Of the suppurating cases some were due to uncleanliness of the patients after the operation, and to their interference with or removal of the dressings, but I think the insufficient toilet of the skin prior to the operation must account for not a few failing to heal primarily. No part of aseptic surgery is stronger than its weakest link, and therefore if a satisfactory result be desired, every precaution before, during, and after the operation must be strictly observed.

The question naturally presents itself, does suppuration affect adversely the desired result of the operation, or may it be regarded

only as the failure of an ideal operation? Most surgeons, I feel sure, regard suppuration as lessening the full benefit of the operation. The scar tissue which is formed as the result of suppurative inflammation, though strong and dense at first, becomes later on less firm and resisting. In addition to this, suppuration often leads to the loosening and removal of the sutures, and this must be a loss to the effective support to the uniting edges of the incised muscles and aponeurosis.

All surgeons, however, do not share this opinion, for on referring to Dr. Dudley Allen's article in the 'Medical Record of New York,' August 1888, I find that he recommends the wound to be maintained aseptic for a week and then made to suppurate, when the silk stitches, as they come out through the external abdominal ring, are removed as they become loose. By allowing the wound to suppurate, Dr. Allen maintains that the inflammatory exudation and subsequent repair provides a firmer and more reliable pad to withstand the constant visceral pressure from within.

As a rule, very little inconvenience to the patient is experienced if suppuration does occur, nor is his convalescence retarded, for the septic suture has often come away or been removed before he has "done his time" of a month in bed. Occasionally months elapse before the suture or ligature is discharged through the sinus, and not infrequently a second operation is needed for the removal of the offending stitch.

Mortality.

The occasional risk to life has to some extent influenced us in recommending the operation for the radical cure, especially when patients express great dislike to the operation. Until recent years we were constrained to confess that the operation was attended with some risk, but under more careful observance of aseptic precautions, danger has so largely decreased that we can give our patients the assurance that there is practically no risk to life.

The results of operations at St. Bartholomew's Hospital during six years ending 1898 shew a total of 650 cases of reducible hernia, with two deaths—one from septic pneumonia and the other from scarlet fever of ten days' duration. Forty-eight cases of irreducible inguinal hernia have been subjected to operation with two deaths, both of preventable origin.

Statistics record that prior to the year 1880 the death rate was largely in excess of that of later years. This immunity is due to the better understanding and carrying out of strict aseptic precautions. The death rate has so steadily decreased that the operation may now be considered a perfectly safe one.

In extreme old age and in infancy the risk to life is considerable, so that the radical operation should not be advocated except under pressing reasons. Kerewsker, in his statistics published in 1891, while strongly advocating the value of the operation in early life, records a mortality of three patients out of 63 cases operated on, shewing a mortality of 4·7 per cent. Mr. Robinson has recorded the results of 64 operations performed for non-strangulated hernia at St. Thomas's Hospital between the years 1879 and 1890, with four deaths, giving a death rate of 6·2 per cent.

Sutures.

A not unimportant detail in the technique of the operation is the selection of the sutures to unite the divided structures and to reconstruct the canal.

The selection of the sutures is practically confined to the choice of four varieties—silk, catgut, fishing-gut, and kangaroo tendon.

Silk is probably the suture most in fashion, and theoretically it fulfils all the essentials of an ideal suture. It can be readily made and kept aseptic, but in my experience—and I know, also, in the experience of others—its use has led to not infrequent suppurative inflammation. Most of us have, at a varying time after the operation—it may be a fortnight or three weeks, or even later—noticed a slight œdema and redness of the skin and subcutaneous tissue, followed by a vesicle which becomes pustular. The pustule breaks and discharges a thin ichorous pus, and in about a week is succeeded by a sentinel-granulation which only too surely indicates a deeper-seated irritation. This is followed by a narrow fistulous track passing down to, it may be, only a buried suture, but not infrequently to a deep undermined cavity involving the entire area of the sutured structures. No healing of this abscess cavity takes place, as a rule, until the suture is removed or comes away spontaneously: this process often takes weeks, sometimes months, to effect.

Dr. Marcy relates that in the discussion upon the use of silk

ligatures and buried sutures in wounds the general verdict was "that aseptic silk, aseptically applied, may be incorporated into the tissues, but remains encysted, and often, after a considerable lapse of time, causes irritation and is expelled as a foreign body."

Mr. Holmes gives it as his opinion that silk sutures fulfil admirably four out of five conditions, the fifth being whether it is so far unirritating as to bury itself in the tissue of an artery and become absorbed or disintegrated without setting up suppuration and coming away. He further says that catgut ligatures are not of trustworthy composition. Ox aorta ligatures are admirable, but the kangaroo tendon ligature presents advantages even over ox aorta.

In a communication to 'Medico-Chirurgical Transactions,' vol. lxix, Messrs. Ballance and Edmunds state they do not consider silk a perfect ligature for aseptic wounds, and they believe kangaroo tendon to be perfectly trustworthy for at least two months.

Catgut.—Various animal sutures have been employed, and among them catgut has its strong advocates. Dr. Marcy, of Boston, U.S.A., appears to have adopted them first in America in the year 1871, but it was not till the year 1878 that he first used it in the cure of a reducible inguinal hernia. Mr. Charles Steele, of Bristol, adopted it in England in 1873 in a boy, aged 8, who remained cured for six months, when strangulation occurred on the same side; herniotomy was performed, and a second curative operation attempted, this time with three sutures in the place of two employed at the first operation. Catgut may be useful as a ligature for the sac, but I think it does not hold sufficiently long, probably not longer than five or six days. It therefore cannot be regarded as a reliable suture for structures needing prolonged apposition and support. This has also been the experience of Dr. Cheever, of Boston, who states that he has tried catgut for the radical cure of hernia, but that it was speedily absorbed and failed. Many surgeons in England and abroad have experienced the same results, and consequently have discontinued its use. On the other hand, there are many who strongly prefer it, and in a conversation I had with Dr. Holloway, of America, about a month ago, he informed me that he always used this form of suture for the operative cure of hernia.

Dr. Martin, of Berlin, is not satisfied with the catgut suture,

for he says :—" I have myself tried catgut sutures, but the results were not satisfactory."

Fishing-gut is regarded by some with favour, but I have discontinued its use, as it does not seem to adapt itself to the continuous form of suture, and the multiplication of rigid knots is, in my opinion, a serious drawback to its use. For the union of the skin incision I nearly always use it, but as an interrupted and not as a continuous suture.

Kangaroo Tendon.—Of late years I have almost exclusively employed this form of suture, the value of which was first brought under the notice of the profession by Mr. Girdlestone in 1882. Its success depends largely on the proper preparation of the tendon, by which it is rendered perfectly aseptic and remains so. For some time I have had it prepared in the following manner :— Each strand is scrubbed, in the same direction, several times with soft soap and water, after which some ether is poured over it, in order to remove all traces of fat. It is then immersed for 48 hours in a solution of perchloride of mercury (1 in 500), and at the expiration of this time it is transferred to a glass jar containing a solution of 1 in 2,000 mercuric perchloride, in which the tendon is kept till required for use. In my experience it has many advantages, for each suture can be made as broad or narrow, thick or thin, according to the varying requirements of the suture. It is, moreover, of sufficient length to allow it to be used, if necessary, as a continuous suture, while it has a great advantage in not being absorbed for many months, thus serving as an effective retaining suture.

Personally I use the tendon as a continuous suture in sewing the muscular fibres of the conjoined internal oblique and transversalis muscles to Poupart's ligament, and also in suturing the incised aponeurosis of the external oblique. The advantage of the continuous suture is the persistent apposition of the edges of the divided muscles. It has, however, the drawback of all continuous sutures—that if any part of it gives way, the whole length gives way too. In the early days of its use at the hospital, when sterilisation was not effective, I had some cases in which suppuration ensued, but since the tendon can be made quite aseptic, suppuration has been of the rarest occurrence. I have only this morning received a communication from Mr. Maxwell, my house surgeon, and he tells me that during the last five

months I have performed 30 operations for the cure of hernia. Of this number 27 have healed by the first intention, and in three superficial suppuration took place. The note adds that in the three cases which suppurated two only were round the skin sutures, and in one suppuration occurred all along the line of the skin wound, and this was probably infected by the pus of boils situated in the region of the incision. In none of the three suppurating cases were any of the sutures discharged, nor were any of the tendon sutures seen.

In private I have only one record of suppuration, and this occurred in a lad with incontinence of urine.

In instances of suppuration the pus formation is very slight, and does not end in the suture coming away before complete healing takes place. In this it compares favourably with suppuration taking place with a silk suture.

I have had no experience with silver wire as a deep suture.

Sex.

Statistics in our Hospital records shew that the operative cure for inguinal hernia is performed much more frequently in males than in females. This disproportion of operation in the two sexes is accounted for in many ways. The male sex up to the age of 20 is ruptured 11 times more often than is the opposite sex. On reference to the tables of the City of London Truss Society, I find that out of 10,771 patients relieved at that institution under 20 years of age, no less than 9,900 were males, and 871 only were females. Among other conditions necessitating the operation in males are their entrance into the public services, their laborious work, and the constant risk of strangulation. In girls the operation is nearly always followed by a successful result, while the social destiny of most of them demands some curative operation. The comparative simplicity of the operation and the absence of embarrassing structures in girls render the obliteration of the canal more effective, while the rapid growth of the pelvis at the time of puberty favours very markedly the success of operative interference in earlier life. The necessity for women to wear some instrumental support for a limited time after the operation is not so imperative as in males.

In men the anatomical relations and conditions of the inguinal structures frequently make the operation one of much difficulty.

The hernia are often large and irreducible, and, especially when congenital, the intimate connection of the vas deferens and the vessels of the cord with the sac render the greatest care necessary, and it is under these conditions that the vas has been incised and in some instances completely divided.

The Use of Trusses after Operation.

It is a moot point among surgeons as to the necessity or advisability for the patients to wear some instrumental support, at any rate for a time after they resume their usual occupations.

No part of the after-treatment excites a greater divergence of opinion than the question of advising the use or not of instrumental support. Some advocate very strongly the necessity of a truss for a time, urging that a tight-fitting truss is important to support the weak spot in the abdominal wall; while others, equally decided, describe the wearing of a truss as positively injurious.

Mr. Ball in the 'British Medical Journal,' 1887, is strongly against the use of a truss, as it tends to produce absorption of the plastic effusion in the canal upon which so much of the success of the operation depends.

Dr. Bull, who has had probably the largest experience of hernia in America, states that he has never seen any harm from the use of trusses. This opinion I accept in all its fulness, for during an experience of over 30 years I have never seen any harm resulting from the use of properly-adapted trusses, while the ground on which the objection is based, that the truss-pressure occasions absorption of muscular and aponeurotic structures, is wholly without warrant. That it may and does cause absorption and displacement of the groin-fat is no doubt true, but this is rather a gain than a loss to the patient.

Personally I always insist on the use of a truss after the operation, and this opinion is, I know, shared by my colleagues at the Truss Society. Its use gives the patient confidence and its pressure is just sufficient to give support to the coapted structures after their division at the time of the operation. This recommendation is the outcome of experience, and statistics show that the majority of the patients who apply for relief are those who have not worn any support after the operation. Of

138 cases of which I have records on this point, I find that 78 were told that they were permanently cured and had no need for trusses, while 60 were recommended to wear them for a limited period. The number of those who were advised to wear a temporary support does not, *per se*, imply failure of the operation, but in the 78 cases a relapse of the hernia had occurred. In private practice I always recommend my patients to wear a light truss for six to nine months.

Conditions which Render the Operation for the Radical Cure Unadvisable.

Operative measures should not be recommended:—

(1) In the case of children under 6 years of age, except under rare circumstances. The large majority of children ruptured from birth, if properly treated, are cured long before this time. Data on this point in general hospital practice are absolutely unreliable, inasmuch as the little patients cease attendance immediately they have had their trusses adjusted, and they gradually drop out of inspection. It is only in private practice that continuity of observing the progress of these patients exists. On reference to my notes I find that over 70 per cent. become cured before the age of 6, so that the question of the radical operation has never to be considered if this be deemed the age before which no operation should be recommended. Whether the hernia in these children reappear in later life I have little knowledge of, for my professional life has not been sufficiently long to know how many recur in later years. Provided these children are carefully attended to, their cure in most cases may be safely predicted. No child in my experience is too young to wear a truss, and precious time is wasted and the cure delayed if an instrument is not applied at the first appearance of the hernia. It is important for their well being that the surgical treatment of these patients should not be relegated to the mechanician, but they should always be under professional supervision.

(2) Patients suffering from organic visceral disease, as diabetes, Bright's disease, ascites, and advanced cardiac or pulmonary diseases, are not eligible for operation.

(3) The size of the hernia does not necessarily contra-indicate an operation, except it is so large that the abdominal cavity is

unable to receive or retain under moderate pressure the extended viscera. If the protrusion be large and incapable of retention by mechanical means a partial operation may be performed which, with the use of a truss, may secure the patient from chance of strangulation.

(4) In very old patients and in most over 45 years of age the operation is not advisable.

(5) Small hernia should not be operated on if the tendino-muscular walls are weak and bulge on coughing over a considerable area.

(6) In the septic peritonitis of strangulated hernia the operation cannot be a complete one as it is necessary that drainage should be employed. Nor should a curative operation be performed if it largely imperils the patient's life.

With these exceptions the operation may be recommended, especially in the case of patients desirous of entering one of the public services, in all cases of congenital hernia, in cases where the hernia cannot be retained by trusses, in many cases of irreducible epiplocele and enterocele, and in uncomplicated cases of strangulated hernia.

Afterwards.

The results of the curative operation for hernia are perhaps the most unsatisfactory part of my remarks, for it is impossible from statistics to obtain reliable data as to the permanent cure of the patients operated on. Difficulties on these points assail us on all sides, and they are not far to seek.

The migratory habits of the London poor and their wanderings from hospital to hospital prevent all periodic inspection and consecutive information. It is not sufficient to accept the patients' statements that they remain cured, for this can only be verified by the personal examination of the surgeon. Patients fail to report themselves from time to time from dissatisfaction at the failure of the promised cure, while many consider themselves cured by reason of ignorance of their condition. The standard of success is variously estimated by operators, some being rigorously critical, while others deem the operation permanently successful if there is no protrusion through the outer ring. The standard of a radical cure, in my opinion,

must be that there is no more bulging on coughing than is present in a perfectly healthy inguinal region.

A large share of the success in these operations depends on the completeness of each detail, and on the sufficient extent of the operation. In many cases which have come under notice the only attempt at cure appears to have been the suturing of the external abdominal ring, which can hardly be deemed a serious attempt to cure the hernia. The results of operations by European and American surgeons teem in our periodicals, but one cannot but be struck with the shortness of time after the operation that the cure is deemed permanent, and the wide differences in the experience of various surgeons is instructive. Thus Tilanus, in 1879, gives a statistical report of about 100 cases with only 10 per cent. of cures. Bassini in his record of 251 cases, followed up for a period varying from nine months to four and a half years after their dismissal from the hospital, states he is able to report that in 239 cases no relapse had taken place, and that in seven only had the hernia reappeared, while in five cases no information was available.

Socin, in his account of patients operated on between the years 1877 to 1880, gives as his experience that 60 per cent. relapsed and 40 per cent. were cured in operations for reducible inguinal hernia, whereas in incarcerated (?) hernia the cures amounted to 90 per cent., and failures to 10 per cent. The periods of subsequent examination ranged from 9 to 43 months.

The records of the City of London Truss Society do not supply any information as to the relative number of successes and failures after operations performed in the various hospitals, inasmuch as we have no knowledge of the total number operated on. Within the last six years 242 patients on whom the radical operation was performed applied for relief, and as probably only 4 per cent. of the ruptured poor in London are relieved at that institution, we may conclude that the number of operations is considerable. Of the 242 patients, 122 were operated on for right inguinal hernia, 89 for left, and 24 for double inguinal hernia; in addition six patients had been subjected to two operations, and one had undergone the radical operation three times.

In private practice the periodic examination of patients is much more complete, for we are able to keep in touch with them for a longer period, and the results are encouraging. With properly selected cases cures will result temporarily, at any rate,

in about 80 per cent., but it is probable that relapses are not infrequent.

"Afterwards," the last word in the heading of this paper, is, after all, the crucial test of the value of our operative work. At present this "afterwards" too often lies buried in the depths of the great unknown from causes over which we have but little control, but which may in the future, without insuperable difficulty, become fully known if the results of continuous inspection were duly chronicled at the different hospitals.

The PRESIDENT said he had felt apprehensive lest some of the cases in which he had attempted radical cure had subsequently found their way on to the list which the author had drawn up of failures. In talking with one's friends and colleagues one was almost tempted to imagine that there was not such a thing as a failure after the radical operation. He thought that surgeons ought to be very careful how they talked about "cures" after this operation. Before students he never talked of the radical "cure," but of the operation *for the radical cure* of hernia. With regard to the hard and fast line which the author had drawn in respect of operations upon children, he himself feared he had operated on children much under 6 years of age. He asked the author what he would suggest in the case of a child of 3 years whose mother failed to look after him, and whose rupture was steadily increasing in size, coming down under the truss every time he cried; if the truss were made to press firmly enough to keep the hernia up, an ulcer formed in the groin. What was to be done? Would the author refuse to operate on account of the child's age? With regard to the material for ligature, he said he had had little experience of kangaroo tendon; catgut he had long since discarded, and he now used silk for everything—interrupted for the closure of the canal, and continuous for the external wound. He thought it was convenient to have the same material for all purposes; his experience with silk had been quite satisfactory, and in only a very small proportion of cases had suppuration occurred.

Mr. STANLEY BOYD observed that it would be very useful if surgeons could learn the ultimate results of their operations. He insisted on the importance of utilising every layer of the abdominal wall in order to obtain the best possible result. The first line of defence was the peritoneum. Mr. Mitchell Banks had likened it to the face of a sea wall: so long as it remained quite smooth and had no crack in it, the waves ran up on it and did no damage, no matter what rubbish had been used behind it; but if there were a defect in the face, the wall would soon go. The first point, therefore, was to get the peritoneum smooth, and the next point was to get it tense. That was where they were likely to fail. It was very common to find a large lax piece of peritoneum inside the epigastric in the adult with an inguinal hernia; one could often draw out as much as would cover the last phalanx of the finger, and unless that was tightened up a pouch or fossa would remain. He was always very careful to deal with this "slack" peritoneum, twisting it up firmly with the sac and placing a ligature on the resulting "rope" as high as possible. When let go, he believed that the peritoneum retracted smooth and tight as a drumhead. He mentioned that some time back he had

had a death in connection with this procedure. He had not realised that it was possible to torsion the bladder into the wound; but he had done so, and had wounded it without any suspicion of what had taken place, and he could not imagine what was the matter with the man until it was too late to do anything to avert the fatal result. The next structure was the transversalis fascia. In a case where there had been a wide neck to the sac it was most important to attempt to close up this fascia by means of a continuous suture. Next came the muscular arch, a point of the very greatest importance. He regarded the normally arising internal oblique as a natural truss over the internal abdominal ring. He urged that every endeavour should be made to reproduce the full origin of the internal oblique, and also the valve-like structure of the canal, by using sutures so as to draw the internal oblique to Poupart over the cord, and the conjoined tendon to Poupart under the cord. He always divided the external oblique up to the level of the internal ring (or beyond this if the pillars were widely separated), sewing it up more closely with fishing-gut stitches. Until lately he used to do nothing with the fat in the groin, and he had often been bothered by the collection of blood beneath the skin; now he always sutured the fat with catgut. He referred to a discussion that he had listened to upon the best materials for stitches and ligatures, and how to render them aseptic. Fishing-gut was often spoken ill of. Some surgeons said that one knot of it had come out, some said two; he himself had seen many reappear. But he did not believe that this had anything to do with sepsis. He used to cut his fishing-gut stitches moderately short, and operated by a straight cut on to the inguinal canal; the two needle-like points of fishing-gut were covered only by scars, and as this stretched they sometimes bored their way to the surface and a small septic sinus then formed. For the last two years or more he had always operated by a flap, and had always put three knots on to the fishing-gut, unless he used it continuously, cutting the threads as close as he could to the knot. Even if the third knot came undone, the ends would not stick out like sharp points, and since he had operated in that way he had never seen a single stitch reappear on the surface. He agreed that if a piece of omentum came down it was best removed, and he himself always drew down as much as he could and cut it off. Some time ago Mr. Bennett had laid it down as an axiom that if one touched the omentum there would be obstinate vomiting, but that had not been his own experience. One point with regard to the removal of omentum, worth noting, was that for two or three days afterwards the patient would have a very tender spot, and often some abdominal pain. He pleaded guilty to having had some instances of suppuration, adding that he knew of no operation in which suppuration gave more trouble than in this. Indeed it was very difficult to know what to do. Every stitch put in to keep back a hernia kept back pus also, and one was naturally loth to remove the stitches. He had seen the pus burrowing beneath and in the abdominal wall, doing great damage, and he always felt sure when this deep suppuration occurred that the patient would be in worse case on discharge that he was on admission. He thought the best plan was to give an anæsthetic, to take out some of the outer external oblique stitches, to pass through the internal oblique and the transversalis parallel to their fibres, and to place a tube in contact with the neck of the sac. With regard to the treatment of the neck of the sac, although he believed it did not make any practical difference, he thought it was theoretically a mistake to transfix it; for this meant that unless the

second, all-embracing turn was carried above the first knots, a sort of pit would be formed towards the peritoneum. Doubtless adhesions obliterated and filled it up very quickly. But with regard to the practice of carrying the ends of this ligature on the sac as stitches through the abdominal wall, he could not but feel that such traction on the sac might very easily produce the depression in the peritoneum which it should be our first object to avoid. The author, he remarked, had referred to the growth of the female pelvis at puberty. What it was that led to the greater frequency of femoral hernia in the female was a point which required reconsidering, since Dr. Arthur Thomson had shewn that the sexual differences in the pelvis were as well marked in a five months' fœtus as at any subsequent period. With regard to the limit of age, he himself had fixed a limit at which the child had obtained command over urination, say at 3. Such children bore the operation very well, and one apparently obtained a large percentage of cures. He admitted that it was very difficult to know what were the ultimate results of operation. That afternoon he had inquired of matron and nurses at his children's hospital how many instances of recurrence after radical cure they could remember, and they could remember one only—the case of a boy of 18 months, with a large strangulated hernia, in which the operation had been somewhat difficult and rather hurriedly performed. A year later the child had whooping cough and blew the "radical cure" to pieces. He was operated on again, and had not been heard of since. He never advised the use of a truss after operation unless at the operation the abdominal structures were unusually weak. At Charing Cross, in adults he had seen very few relapses out of, perhaps, 300 cases—always excepting cases of deep suppuration and a few bad cases in which operation was undertaken only to enable a patient to wear a truss.

Mr. Osborn asked how long the author thought it necessary for the patient to wear a truss after operation. He himself always advised a truss for six or nine months, though patients did not like the idea because they had anticipated being enabled to dispense with a truss after the operation, and that was a disappointment. He regarded it as a precautionary measure to assist in the consolidation of the wound. In children under 6 the constant wearing of a truss would often determine a cure without any operation. He agreed that it ought not to be called a radical cure in view of the number of cases that turned up at the surgical appliance societies in which the operation had proved a failure.

Mr. Langton, in reply to the President, stated that most of the London hospitals were represented in the list of failures for the radical cure at the City of London Truss Society, but up to the present he failed to find the President's name amongst them. He had advocated in his paper the use of the kangaroo tendon, but he did not wish thereby to depreciate either the silk or fishing-gut suture. Although he placed the age of 6 as the earliest limit for the operation, there must occur from time to time instances in which it was advisable to operate at an earlier age. He thought the President had adduced a somewhat extreme example of a hernia necessitating operation, but he wished to lay down some elastic conditions under which the operation might with propriety be advocated. By placing the age limit at 6 he excluded practically the necessity of any operative procedure in infants, as if they were properly advised and looked after, the hernia would, in the large majority of cases, be cured long before that age. It was by no means an unusual occurrence for the hernia never to be seen after the adjustment of the first truss,

and no surgeon would suggest the performance of an operation for the cure of a hernia which had not come down for months or years. On the whole he thought this age limit not a bad one. In replying to Mr. Boyd the author stated that in three instances patients of his had presented themselves for failure of the operation, but in two they were advised by Mr. Boyd to procure some support as the results were not quite satisfactory. He concurred that one of the most important details was the dealing with the sac, and in consequence of his experience he advocated the operation recommended by Ball of twisting the sac as tightly as possible, so that it would have a convex prominence at the inner ring, and thus act as a solid buffer. He had made many experiments on the cadaver to prove the existence of this buffer, and he found a more or less convex boss on the peritoneal aspect of the inner ring. He thought the objection of a cavity being left at the inner ring after the ligatured sac had been reduced was more imaginary than real, and, indeed, if a cavity did exist, no harm resulted. Mr. Boyd had further alluded to the bladder being occasionally included in the ligature, and Mr. Langton expressed the opinion that this calamity might occur to any surgeon; but to lessen as far as possible such an accident he always opened the sac and passed his finger into the peritoneal cavity. He laid much importance on strongly suturing the arched fibres of the internal oblique and transversalis to Poupart's ligament as far as the external ring. Not an unimportant part of the treatment of hernia in children consisted in well-directed gymnastic exercises, as they tended to increase the development and the retentive power of the abdominal muscles. He had not discussed the subject of femoral hernia as being outside the scope of his paper. He pointed out that femoral hernia was comparatively rare in girls under puberty, but more common than inguinal during the child-bearing period, while, after the menopause, inguinal hernia was again in the ascendant. If, however, a woman developed a hernia whilst pregnant, the protrusion was nearly always inguinal, in the proportion of 99 per cent. of inguinal to 1 per cent. of femoral hernia. He was glad to gather from Mr. Osborn that he advised the use of some instrumental support for a limited period after the operation. Trusses in infants and children were easily adjusted and retained in position, and if excoriation resulted it was nearly always due to want of proper attention or to the mal-position of the truss.

March 27th, 1899.

DEATH FROM FUNCTIONAL NERVOUS DISEASE.

By Robert Maguire, M.D., F.R.C.P.

We are well acquainted with death occurring in the course of a functional nervous disease, but such death is usually due to some intercurrent affection to which the primary disease predisposes the patient. Thus death may occur in the course of acute mania or of the status epilepticus, from simple asthenia, the result of the riotous action of the body powers. Anorexia nervosa, and allied hysteric states, may so weaken the body from the inability to take nutriment as to kill by simple asthenia, or by leaving the patient unprotected from tuberculosis. Again, death may occur from concussion of brain. Those who have made many *post-mortem* examinations on such cases cannot believe that the death is due to the often small amount of contusion or laceration of brain substance which possibly is found. Sometimes no such laceration is seen, often a much greater laceration is recovered from or gives rise to no serious symptoms, and further, Mr. Horsley has shown that death in such cases is due to disturbance of the respiratory centre and not to the local brain contusion. We know, too, of deaths from fright, the heart's action being suddenly stopped; and such, I believe, is not uncommon as the cause of death in sudden hæmoptysis. Lastly, death from shock, primarily affecting the functions of the sympathetic nervous system, is indirectly caused by vasomotor paralysis.

But the cases I wish to bring before the Society this evening are not of this class at all. I have avoided calling them neurasthenia; they are not deaths in the course of functional nervous disease, but deaths actually *from* functional nervous disease—that is, without disturbance of any one particular nerve centre or other somatic function, but (to anticipate what I shall have to say of their pathology) they are deaths from simple exhaustion of grey matter. As I should like the Fellows to bear this in mind while listening to the description of my cases, I must premise that the

cause of the exhaustion may be of variable power. Grey matter which is originally strong will require a strong force to exhaust it, but that which is already weak may have its force destroyed by but a feeble cause.

In March, 1888, there came to my out-patient room at St. Mary's Hospital a married woman, aged 52, who had been seen on the previous day at Moorfields Hospital by my colleague, Mr. Silcock. Nearly three months before she had had a fall and hurt the back of her head, and gradually, she said, her eyesight began to fail, until she went to the Ophthalmic Hospital because of almost complete blindness. Mr. Silcock sent her on to me for a medical opinion, but without telling me anything of his finding or of his views of the case. She told me that she could just tell lightness from darkness, but could distinguish no individual objects. Her pupils contracted to light, and as there was no voluntary accommodation one could not ascertain the behaviour of the pupils during this exercise. There was no inequality of pupils and no strabismus. The patient complained of pain in the back of the head and of dizziness on standing. On trying to walk she staggered somewhat and seemed inclined to fall, usually to the left, but I noticed that she avoided the chairs and tables in the room. Her reflexes were normal, and on examination of the eyes with the ophthalmoscope I could find nothing abnormal. There had been no vomiting or convulsions and her general health was good, her pulse of normal tension, her urine contained neither albumen nor sugar, and all her organs seemed healthy on examination. She had previously suffered from hysterical attacks. I came to the conclusion that the case was one of traumatic hysteria, and found afterwards that this also was Mr. Silcock's opinion. I admitted the patient into the hospital for further examination, and two days later she was seen by one of my senior colleagues, who diagnosed tumour of the cerebellum. Two days later still, the resident medical officer summoned me to see her, for during the night she had become unconscious. I found her, at about 10 a.m., lying on her back, breathing quietly, her pulse of normal tension, having a frequency of 80 in the minute, and her reflexes were normal. The eyelids contracted when the conjunctivæ were touched. The right pupil was larger than the left, both were insensible to light, and the right side of the head, face, and neck were bathed in perspiration while the

same parts of the left side were dry. The limbs fell flaccidly after being raised. The patient could be roused to say "Yes" or "No," incoherently in answer to questions, and, when asked, tried to put out her tongue, but was otherwise unconscious. Her temperature was normal, the urine and fæces were being passed involuntarily. At this time I altered my diagnosis and agreed with my senior colleague that the case must be one of cerebellar tumour. She died on the evening of the same day. Two days later I made a *post-mortem* examination and macroscopically, as well as by a most careful microscopic examination, nothing whatever abnormal was found in any part.

Of the second case I have to mention, I have but few details. A young woman, aged 26, unmarried, who had suffered no injury, was admitted into St. Mary's Hospital in an unconscious state. The history given was that after a long-continued trouble she had for a few days complained of vague pains and seemed queer in her manner. Afterwards she was unusually drowsy, and finally became unconscious, in which state she was brought to the hospital. On admission her temperature was normal, her pulse in frequency and quality normal, her urine drawn by the catheter normal, and her breathing of normal frequency. Her pupils were equal, of ordinary size, and contracted to light, and the conjunctivæ were sensitive to the touch. Her urine and fæces were passed involuntarily. The usual reflexes were present in normal degree. She was simply unconscious, and could not be roused. In this state she lingered for four days, and then died. There was nothing to indicate poisoning, and once more on careful *post-mortem* examination I could discover nothing abnormal, either by the naked eye or by the microscope.

The third case is one upon which I had opportunity of long observation and is also remarkable in that recovery took place from one attack of the symptoms, followed some years later by a second attack and death. The patient was one of my fellow students in Manchester, and though junior to me in the school had begun the study of medicine late in life. His previous history is of importance. He was a Scotchman and had been a schoolmaster. He had undeniable abilities, and had taken the B.A. degree of the London University, but his abilities were distinctly those of a pedagogue, and I have sometimes noticed that such powers seem to be incompatible with the development

of a medical mind. He found great difficulty in mastering the subjects of the medical curriculum for the ordinary diploma. Moreover, as I found afterwards, his means were very straitened, and he had for this reason during his studentship quietly existed upon a diet which was insufficient to maintain an active brain. Yet by dint of hard work, perseverance, and great self-denial he succeeded in passing his examinations and was unduly elated at doing so. About three weeks afterwards I was summoned to see him and found him unconscious, breathing labouredly but not stertorously. I heard that he had complained of posterior headache and sleeplessness for a few days, and was found one morning in the condition in which I saw him. His limbs were flaccid, and irresponsible to stimuli, excepting that the knee-jerks and the plantar reflex could be obtained in normal amount. His pulse beat was slightly increased in frequency, but the arterial tension was normal. The urine and fæces were passed without warning and the urine withdrawn by the catheter was normal. There had been no vomiting. There was no strabismus, the pupils were equal and of ordinary size, and reacted to light, and on ophthalmoscopic examination the fundus oculi on each side showed no change. The patient would occasionally roll from one side to the other, but generally lay upon his back, and could not by any means be roused from his unconsciousness. The temperature was raised for some four days, but never exceeded 100·5°, and afterwards was normal. Such was the patient's state when I saw him first and so it remained for nearly three months, with only two variations. At one time there was obstinate constipation relieved only by an enema. Again on one occasion there occurred an epileptic convulsion, due to careless overfeeding, for which I was, I am afraid, responsible. In the early days of the disease the patient could swallow no solid food, only fluid would pass down the œsophagus, and I told the friends to feed the patient on milk and beaten-up eggs and give as much as they could. This was right enough at the time, for but little could be swallowed, but later on my orders were obeyed literally, and I carelessly did not inquire how much food was being given, satisfied by hearing that food was being taken well. After the fit, I was horrified to find that no less than six quarts of milk and 12 eggs had been administered in 24 hours. This was the cause of the epilepsy. After this I need scarcely say that the patient showed no

emaciation during his prolonged illness. I at first could form no diagnosis of the case, but at that time there appeared the first edition of Sir Samuel Wilks' 'Lectures on the Nervous System.' I was struck with the resemblance of the symptoms to those described in that work as due to encephalitis. At this period I was only beginning my work as a physician, and in my inexperience assumed that encephalitis was the disease my fellow-student was suffering from, that after so long a duration of the affection his brain must be almost in a state of pulp, and that recovery was impossible. Professor Dreschfeld, who saw the case with me, with a more mature experience refrained from giving any diagnosis or other opinion, and was justified in the result. At the end of about three months the patient gradually awoke—there is no other description to be given of the process. He gradually recovered all his powers and to all seeming became quite well. He began practice, married, and continued well for some three years. Then, as I am told, for I had left Manchester and he was not at this time under my observation, the same symptoms recurred after a period of overwork and anxiety. He was admitted into the Manchester Royal Infirmary in a condition the same as that in which I first saw him, but he had no rise of temperature. The late Dr. Ross then thought his state analogous to that known as *melancholia attonita*. He died in about six weeks, and nothing whatever abnormal was found by the pathologist at the *post-mortem* examination. The patient's family was not neurotic; on recovery from his first attack he told me that he had never put himself in the way of contracting syphilis, and I learnt from his friends that he was a very religious man, and had led an absolutely irreproachable life.

My fourth case I would specially call attention to, because, by reason of my experience of the former cases, I was able to diagnose the condition during life, and this diagnosis was confirmed, as I think, by *post-mortem* examination. Some three years ago I was called to see in consultation, a few miles out of London, a retired clergyman, aged 62 years. He came of a neurotic stock, and I should judge from accounts had been of somewhat eccentric habits, though of undoubted great mental ability. Being possessed of ample means, he had given up the active service of the Church, and lived more or less as a recluse, devoting himself to literary studies. He had had serious troubles.

Always somewhat taciturn and retiring, he had been unusually silent for some days, though otherwise in apparently good health, when one morning he was found to be unconscious, and could not be roused. The urine was withdrawn by catheter, and found to be normal. When I saw him, I simply found him unconscious and incapable of being roused in any way. His pulse was of normal tension, and without entering into details, as I have done in the previous cases, I may simply say that I could find nothing whatever abnormal with any functions, except those of the nervous system. To all intents and purposes the man was simply asleep, and could not be awakened. I ventured on the diagnosis that this condition was due simply to failure of the grey matter of the nervous system, and upon the prognosis that the man would die. He did die the next day and, obtaining permission to make an autopsy two days after death, I found, in short, nothing whatever which was abnormal, macroscopically or microscopically, in any part.

There will be in the recollection of the Fellows a *cause célèbre*, which occurred some seven years ago at the Rugby School. A little boy, not suited to such severe exertion, was running in a paper-chase. He had been in good health. After running for some time he suddenly fell in the middle of a field, but quickly picked himself up and ran on for about two fields farther, when he dropped once more, was picked up unconscious, and died next day. I need not here enter into details of the case, for they are published in the 'Royal Medico-Chirurgical Proceedings' for May, 1893. At the reading of the paper on the case, many suggestions were made as to the cause of death, such as, that it was due to undue presence of sarco-lactic acid from muscular exertion, to heart failure, to the accumulation of ptomaïnes, to dissolution of the blood corpuscles, and so on, but I think it is no unfair criticism to say that the discussion was futile, with the exception of one remark, which passed almost unnoticed. My friend and colleague, Dr. Waller, the physiologist, was sitting by me at the meeting, and near the close of the debate suggested that the death was due to failure in power of the grey matter of the nervous system, the result of overwork in the nervous effort required to keep up in the running when the body was incapable of such exertion. Dr. Waller's remarks were short and hurried, and probably for this reason were not properly understood by

those present. I confess that, in private conversation, I rather jeered at him for his idea, but further explanation from him, and consideration of his view by myself, convinced me that he was right.

The cases I have described to-night are, I would maintain, instances of the same pathology. In the first case mentioned an injury was the immediate cause of the nerve exhaustion, and acted upon a nervous system already predisposed to lose its power. In the second case, incomplete as its account is, worry and trouble had worn out the grey matter. In the third, mental work, which in a properly constituted individual would have been but a normal exercise, was too much for a brain unsuited for such studies, especially when enfeebled by anxiety and lack of ordinary nutriment. In the fourth case, a constitutionally ill-balanced nervous system exhausted its energy. In the case of the Rugby boy quoted, the strain upon the grey matter involved by determined "pluck," and determination to keep up in the paper-chase, exhausted its powers.

I have not been able to find anything in medical literature which bears directly upon the phenomena of these cases, though possibly such may be recorded under headings which render it difficult to trace. Nor do I find anywhere mentioned the point of pathology to which I now draw attention—death from exhaustion of grey nervous matter. Yet I believe it is a pathological fact. Clinically it is characterised, I would point out, by the uniform and equal failure of all the body functions. Outside the nervous system there is no picking out of one particular system which shall give way before the rest, as occurs in all other general diseases. Even in the most gradual and equal of all other deaths, namely, that by senile decay, some one system fails more than the others, and most frequently is the failure shown by the cardiac muscle, since this can have no resting time for recovery. But in the cases described, the organic functions were equally enfeebled to the last, and we are therefore driven to the conclusion that the seat of the disease must have been in some part which governs all the others. That part is the grey matter of the nervous system—not to particularise its more exact situation. This is the most highly evolved, and last-developed mechanism. Without its control all the other systems would fail to work smoothly and equally. Such a part is by its position and development more

prone than any other to suffer from general lack of vitality, and I would maintain that the cases I have described are instances in which its exhaustion has led to stoppage of all the other vital functions.

Minor instances of such disorder are common enough, as we all well know. In the form of hysteria and neurasthenia we are prone to almost ridicule them, but such cases as I have described illustrate how very serious at times the grey-matter exhaustion may be. I think, too, if we recognise and admit the pathological view mentioned, we have offered to us an explanation of many extraordinary cases which usually are told only as curiosities. We all, whether surgeons or physicians, are awake to the importance of what we call "depressing surroundings." We know that however perfect may be the other hygienic conditions, yet worry of any kind, especially companionship which is actively or passively irritating, will delay the healing of a wound, or the resolution of a pneumonia. This, I think, is the effect of exhaustion of grey matter in a minor degree on the vitality of the body functions. The sense of impending death is due to a similar exhaustion, and its importance depends upon whether the grey matter is capable of recovering itself or not. Thus Murchison in this respect draws a distinction between typhus and typhoid fever. In typhus, a severe but short fever terminable at a definite date by crisis, such a sense is of little importance, for the strain upon the nutrition of the grey matter will be soon over and the patient immediately after the crisis rapidly recovers his assimilative powers, his nutrition, and his strength. But not so with typhoid fever. There the sense of impending death carries with it a most serious prognosis, for the fever is long, the termination is by lysis, after which a long period exists before the assimilative powers and nutrition recover themselves. Thus the grey matter tends to become more and more exhausted as time goes on and its governing force on the organic functions is wanting. Again, the sense of impending death in young persons can be laughed away, for their grey matter has recuperative power. But the opposite is the case when the same morbid feeling occurs in aged persons; with them more often than not it is a sure harbinger of the end, for recuperation is impossible. In regard to this matter the effect of nationality is interesting. The adults of Eastern nations may be considered the equals in

vitality of the aged of European races, and our doctors who have practised in the East tell us of how frequently an East Indian says he is going to die, and without any ascertainable physical cause he simply lies down and does die. He has no reserve of vitality and the first symptom of failure of his grey matter is important. On the other hand I think I might point, as a contrast among Western nations, to the Irish race. An Irishman's illness is always by his own account most dangerous. There may be some here who like myself have attended an Irish woman in labour and can bear witness to the plenitude of her complaints and alarms. But the Irish illnesses can *cæteris paribus* bear a favourable prognosis, for, as I have on a previous occasion pointed out to this Society in discussing the question of gout, the Irishman has more reserve of vitality than almost any other man. Neurotic as he undoubtedly is, he is yet the exact opposite of the Eastern, and has recuperative power to possibly its highest extent.

We have at present very few data upon which to ground views on the question of exhaustion of grey nervous matter, nor can we have until the science of experimental psychology has made further progress, and in England but little has as yet been done in this direction. I would refer my hearers to an article by Dr. Waller on "The Sense of Effort: an Objective Study," published in 'Brain' for 1891. This deals especially with nerve exhaustion in its relation to muscular exhaustion, and its bearing upon the case mentioned of death while running will be obvious. But there are matters mentioned in that article which bear strongly upon the points mentioned in this paper, as to the general exhaustion of central nerve matter. Thus: "Voluntary fatigue is in major part central, in minor part muscular"; "fatigue, the consequence of effort, manifests central as well as peripheral objective signs." Let these remarks be read in connection with what I have premised, that the cause of exhaustion of grey matter may be of variable force, according to the primary ability of the grey matter for action, that is, its force of vitality. We then see how it is possible for a strong grey matter to be exhausted by a strong effort, a weak grey matter by an effort scarcely appreciable, the effort in each case being nervous, but the common result must be the manifestation of "peripheral objective signs," which in the cases I have mentioned have led to

death. I must, however, explain that to take such a view is to go beyond exactly what Dr. Waller states in his article, for he deals especially with the co-ordination of central (nervous) and peripheral (muscular) fatigue. But a consideration of his article gives one a clue to the otherwise inexplicable deaths which I record. Moreover, it would be easy, did time allow, to show how many other cases met with clinically, but not ending fatally, are not only explicable, by the view mentioned, but strongly support it. My thesis, however, is that exhaustion of grey nervous matter may be so great as to cause absolute death of the whole body, and that the existence of such a condition gives rise to characteristic clinical phenomena, which may lead to the recognition of the diseased state before death has occurred.

It must be noted, however, that I have spoken only of "exhaustion of grey matter," without any argue as to its more specific cause, beyond that, in the cases I have mentioned, there seem to be eliminated any of the ordinary auto-intoxications, and also any of the poisons which might be introduced from outside. The point I lay most stress upon is that the symptoms of somatic failure have been equal and gradual, except in so far as concerns the nervous system, and to this system I look solely for elucidation of the cause of death. But it is possible that the nervous system itself may by overaction, or irregular action, provide its own poisons, on the same principle upon which certain chemical actions are stopped by their own products. Further investigation upon this subject also is necessary, and is progressing. I would refer the Fellows of the Society to the researches of Drs. Halliburton and Mott upon the pathological products of the cerebro-spinal fluid and the effects of choline and neurine on nerve-tissue, but more especially to the experimental observations of Dr. Waller upon the action of these substances, and also of muscarine, snake poison, tetanus toxin, and atropine upon the isolated nerves of the pike and the frog. Time will not allow me to do more than merely mention those researches, but their results are very important on this question of exhaustion of grey matter, and, I venture to think, seem to support the view I have given in explanation of these strange cases. If such a case were again to come to me, and die, I should certainly endeavour to ascertain by the assistance of others skilled in such investigations what might be the composition of the cerebro-spinal fluid.

Dr. Sansom criticised the use of the term "functional" as applied to such cases. He could suppose patients with ill-defined, strange, possibly serious symptoms; some did not get any better and ultimately died, but the morbid anatomist found nothing. One might find a similar history in the past in the matter of amaurosis. There was a time when this term comprised a large number of conditions in which, as a German professor once observed, neither the patient nor the doctor saw anything. The obscurity in that regard had mostly passed away, but the obscurity in respect of these deaths remained. He inferred that there was a peculiar condition of irritable weakness or some morbid state of the grey matter. He pointed out that an analogous state of things existed elsewhere in pathology, as in epilepsy in a child; the child died and the morbid anatomist found nothing. The same thing occurred in the adult in angina pectoris, not even atheroma of the coronary arteries being always present. He recalled that the late Dr. Arnold died from such an attack, but he had only one coronary artery, and it was very likely that the cause of death was ischæmia of the heart. Every case such as the author adduced must be analysed. In the first case there were some strange vasomotor symptoms, for instance hemi-hydrosis was noted. That must mean a unilateral affection. In some other instances they might reason from analogy, and he recalled the case of Colonel Townsend, who, by the exercise of a voluntary power, produced a distinct effect upon the cardiac function in the direction of diminishing its pulsations. That case showed that there were some strange and subtle circumstances and conditions of the nervous system from causes which were very difficult to find out. He discussed the possibility of arriving at a working hypothesis, and observed that towards the end of the paper a little of the veil had been lifted when the author said there was a feeling as if the patient were going to die. That feeling must mean something. With reference to the particular nervous area affected or likely to be affected in such a case, he thought it must be that of the vagus, which was the nerve of comfort of the heart and in part, at any rate, probably presided over its nutrition. In any case he thought that the primary effect was upon the area subserved by the vagus. Inasmuch as there were some diseases of the nervous system of exceedingly obscure pathogeny, but which, nevertheless, owed their existence to some subtle imported poison, so, in some of these cases, a state of irritable weakness might have been induced in part of the grey matter, and consequently the vagus probably became impaired in its function, and life became extinct. He suggested that many cases of supposed fatty degeneration of the heart died from some obscure nervous cause of the kind alluded to by the author; fatty degeneration of the heart was a rare cause of death. He believed then that the cause of death in these cases must narrow itself to the area of the vagus and not involve all the grey matter; secondly, that there must be a cause, probably a state of irritable weakness, brought about by some somatic cause; thirdly, it explained, possibly imperfectly, some of the sudden deaths which were attributed to fatty degeneration of the heart.

Dr. Seymour Tuke said that some of the cases reminded one very much of the seizures met with in general paralysis of the insane. He had seen men lie in very much the same condition as the woman who had hemi-hydrosis, *i.e.*, in a state of semi-unconsciousness for seven or eight days or even longer; a condition in which they could just be roused, when they would attempt to put the tongue out, and seemed sensible to a certain amount of external irritation. Later on in general paralysis one met with

an extraordinary emaciation in spite of feeding. He asked whether the author had had the temperature taken in his cases, and he referred to two cases of general paralysis, in one of which the temperature before death was over 108, and in the other 107.

Mr. Peeke Richards commented on the fact that in the author's four cases no pathological condition was found after death. He pointed out that in two or three of these cases death had not been very rapid, and if these serious symptoms were due to alterations in the condition of the grey matter there was time for some trace of degeneration to be apparent in the cells in these layers of the cortex. He did not think the cases in which no microscopical pathological appearances were discovered agreed with cases of general paralysis, for in these marked changes in the cells of the cortex were usually found. He could not but think that there must have been some apparent change in the grey matter after death in these cases.

Mr. Battle referred to the case of a woman, aged 30, who was admitted to St. Thomas's Hospital for some dyspeptic symptoms under Dr. Payne, and who, suddenly one evening, was seized with symptoms supposed to be due to perforation of a gastric ulcer. He saw her with a view to operation, but came to the conclusion that this diagnosis was not the correct one, although it was very difficult to say what it really was. She became collapsed with severe epigastric pain, and vomited once, the temperature falling to 97°. She lived about three weeks with symptoms of varying character chiefly referable to the stomach. *Post-mortem* there was absolutely nothing abnormal to be found anywhere in the body.

Dr. Maguire, in reply, said that by "functional" he meant a condition in which no anatomical evidence of disease could be found. He would prefer to style the paper "Death from exhaustion of the grey matter," but this, he admitted, was open to contention. Little or nothing was known about the subject except by those who had studied experimental psychology. He had purposely been vague in regard to the part of the nervous system involved, but he was unable to agree with the view that it was only a question of vagus paralysis, for that would not explain the unconsciousness, for example. He himself held that there was loss of vitality on the part of the last and most specialised tissue in accordance with the law laid down by Hughlings Jackson. He said he was not surprised to hear it suggested that these cases resembled general paralysis of the insane, but in three of his cases the temperatures were not raised, though in one it went up to 100·5 during the first attack for a few days, and in the second it remained normal, differing in this respect from the so-called congestive attacks of the general paralytic. He maintained that general paralysis was not a definite disease at all, but merely a collection of symptoms. It ought to have a much wider application than lunacy physicians were apt to suppose. It was simply an involution of the highest powers of the nervous system. There was absolutely no microscopical or macroscopical change in his cases that he could find. The first case was unconscious for two days, the second for four days, and the last for two days, but the third was unconscious for three months in his first attack, and recovered apparently perfectly, so there could hardly have been any organic change. Even after his last and fatal illness no change could be discovered.

CERTAIN POINTS IN THE PATHOLOGY OF THE PERICARDIUM.

By Harold L. Barnard, M.S. Lond.; F.R.C.S. Eng.

When a large quantity of venous blood is driven into the right heart, the pericardium supports the heart so as to prevent over-distension. It prevents dilatation beyond a certain point at which the walls of the heart are thick enough and strong enough to empty themselves at each recurring systole. The pericardium bears to the heart muscle the same mechanical relation as the leather case does to the inside case of a football, or the outer case to the inner case of a Dunlop tyre. As I believe this observation on the function of the pericardium to be original, you will perhaps allow me to demonstrate it. (The full account of the experiments will be found in the 'Proceedings of the Physiological Society,' March 12th, 1898.)

The pericardium is a very strong and inextensile membrane. Strips of it rolled into cords will bear a very great strain without stretching or snapping. If an entire pericardium is inflated by a bicycle pump it ruptures at a pressure of from one and a quarter atmospheres to two atmospheres, which is many times greater than arterial blood-pressure. It stretches very little before it ruptures. The pericardium limits the capacity of the heart by about one half. The excised heart of a cat in its pericardium was squeezed empty and then distended with saline solution at about a venous pressure. The amount required to fill it was noted and the pericardium was then freely opened. The heart then took as much saline again before it was distended. The extra quantity of saline was mostly received by the right auricle and ventricle. Experiments upon frogs and cats and dogs—the latter animals under anæsthesia—showed that in these the pericardium supported the pulsating heart, especially the right heart and during diastole. The heart was supported not only when a large quantity of blood was forced into it, but also when it was passively dilated by pushing the chloroform.

We must now consider the circumstances which lead to such engorgement of the right heart that it is necessary that it should

be supported by the pericardium. In the first place every contraction of the muscles of the limbs will flatten the deep veins and suddenly inject into the superior or inferior vena cava, and so into the right heart, a very considerable quantity of blood. The venous blood is returned from the legs against the action of gravity by this pumping action of the skeletal muscles of the legs. The most frequent and powerful cause of sudden engorgement of the right heart with blood, however, is the active working of what has been called by Dr. Leonard Hill and myself the respiratory pump. The object of this pump is to raise the blood from the capacious sinuses of the abdomen and when the vascular flow is rapid, as it is during active exercise, each stroke of this respiratory pump will drive a considerable wave of blood through the heart.*

We must pause for a moment to consider this respiratory pump and the important action of the skeletal muscles in carrying on the circulation. The researches which have been carried out by Dr. Leonard Hill and by myself would seem to show that in man the heart cannot carry on the circulation alone for any length of time in the erect position—that the action of the skeletal muscles is necessary to secure the return of venous blood from the legs and the abdomen. This is illustrated by the death of people who have been crucified. After a day or so œdema of the feet appeared; this spread until the heart was so depleted of fluid that it could not supply the brain. When hutch-bred rabbits are held up by their ears for five or ten minutes the blood accumulates in their abdomen to such an extent that their brains become anæmic, their corneæ become insensitive and their pupils dilated, and they soon die.† In man the heart is a pump which is situated above its reservoir or well, the abdomen (*see* Fig., R.H.). At each relaxation it must fill itself from the great veins placed vertically below it. In all such pumping arrangements there are two means of raising the required fluid—either by sucking it up from above or by driving it up from below. Both these methods are employed to keep the heart supplied with venous blood. Every inspiration produces a negative pressure in the thorax (*see* Fig., 2) and therefore in the right heart, and venous blood is sucked from the abdomen into the heart and is driven at each systole into the

* 'Journal of Physiology,' 1897.

† Dr. Leonard Hill, 'Proceedings of the Physiological Society,' 1898.

lungs. This suction pump is all-sufficient during rest and quiet exercise, but during violent exercise and exertion, when the full flood of the circulation is pouring through the dilated arterioles of the muscles and the stroke of the heart pump is both deeper and quicker, the blood must be lifted more rapidly and supplied to the heart in fuller volume. It is then that the abdominal force-pump comes into action.

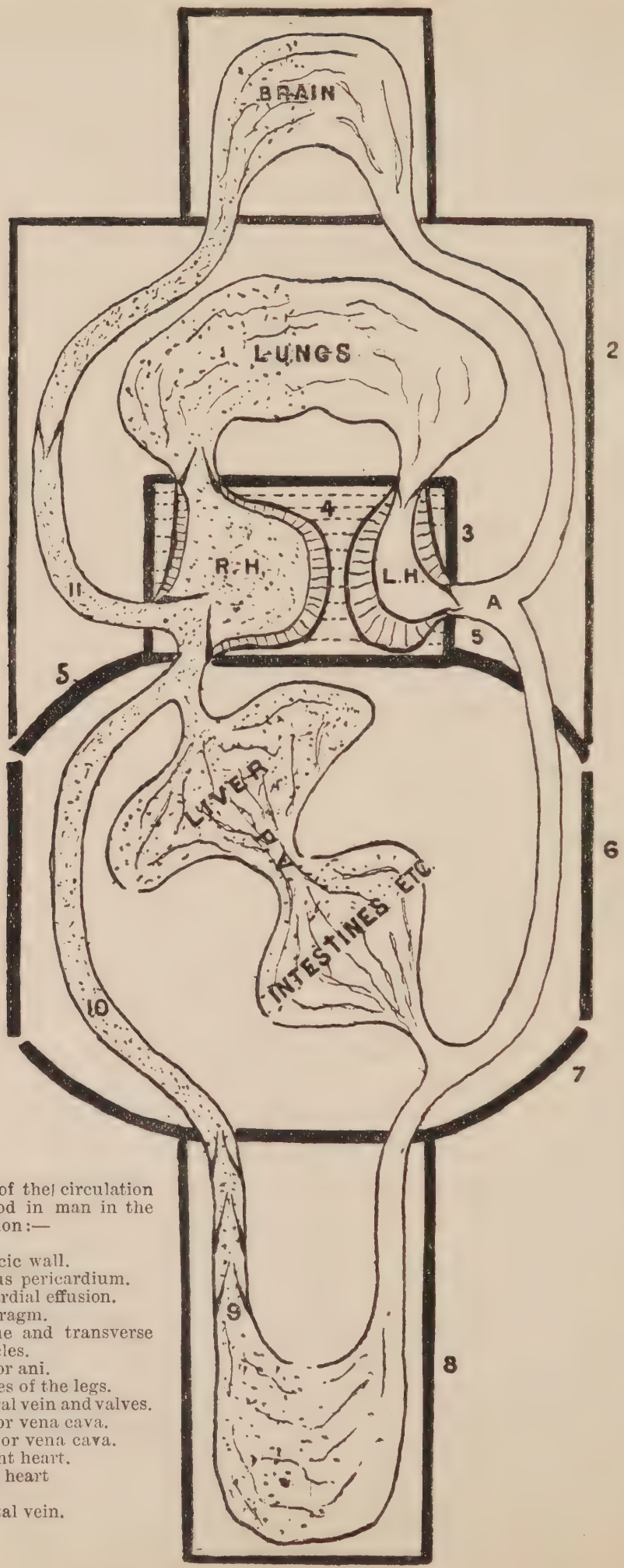
When we consider the anatomy of the abdomen we find that the viscera are contained in a muscular sphere (*see* Fig.). Above is the dome of the diaphragm, and below the basin of the levator ani, closing the outlet of the pelvis. Its walls are formed in front by the recti and behind by the quadrati lumborum muscles, and at the sides by the oblique and transverse muscles. Its veins are large and flaccid and capable of containing all the blood of the body were they not supported by this muscular sphere. During vigorous exertion this sphere of skeletal muscle contracts with each inspiration and drives the blood from the great venous sinuses through the aperture for the inferior vena cava in the diaphragm, directly into the right heart. As this occurs at the same time as suction is exerted from the thorax the maximum flow of venous blood is effected. When we consider that the limbs and the abdomen frequently contract simultaneously and violently in such exertions as running or lifting weights, it will at once be perceived that a large quantity of blood, probably amounting to one or two pints, may be forced into the heart in one huge gulp. It is under such circumstances that the pericardium prevents over-distension.

The pulmonary circulation during vigorous exercise is in a very large degree carried on by the respiratory movements. The pericardium supports the heart and prevents it dilating, whilst the abdominal muscles empty the venous sinuses of the abdomen through the right heart into the lungs. This is assisted by the coincident inspiration of the lungs which enlarges their capacity for both air and blood. During the ensuing expiration the lungs are squeezed empty like a sponge into the left ventricle, for the way to the right heart is barred by the pulmonary semilunar valves. The flow of blood through the liver is also brought about by respiratory movements. The descent of the diaphragm in inspiration squeezes the portal vein and empties the blood through the liver into the thorax. It seems probable that the

great veins of the abdomen store a reserve of blood during rest and sleep, the muscular sphere containing these veins relaxing to a corresponding degree. Directly the person awakes or resumes active exercise the tonicity of the abdominal muscles is increased, and this reserve of blood is driven into the general circulation and distends the heart and the arteries and veins supplying the active muscles.

We will now turn to certain points in the pathology of the pericardium and of the mechanism of venous circulation of which it is a part. When the pericardium becomes inflamed it becomes softened, and we may infer that any sudden exertion or effort, especially a cough, will distend the heart with blood and cause it to press against the pericardium and so dilate it. I believe that a pericardium so dilated cannot recontract—that the heart within it will not in future receive support when it becomes engorged with blood, and that every effort will throw the heart out of gear, dilating its cavities and rendering its valves relatively incompetent and so producing cyanosis and breathlessness. I would therefore suggest that when the pericardium is inflamed the most absolute rest should be maintained not only until the inflammation has subsided, but until the inflammatory materials have organised into firm fibrous tissue. It appears to be of the greatest importance that pericarditis should be recognised early, for if the child be allowed to run about the pericardium may be irretrievably dilated. To return to the comparison of the pericardium and heart to the outer and inner case of a Dunlop tyre, if a hot day softens the outer case so that it bulges under the pressure of the air within we may patch it for a while, but it is irreparably damaged and dilated and goes progressively from bad to worse.

We will now consider one or two points about pericarditis with effusion. At first sight it appeared a most difficult problem how the effusion could exert sufficient pressure to dilate so tough a membrane as the pericardium without collapsing the thin auricles and preventing the ingress of venous blood. It was, of course, certain that venous blood must be introduced into the auricles at a higher pressure than that of the pericardial effusion or it could never enter at all. It was equally certain that this pressure must be high or it could not dilate so inextensile a membrane, but how this high venous pressure was obtained did not



Schema of the circulation of the blood in man in the erect position:—

1, Skull.
2, Thoracic wall.
3, Fibrous pericardium.
4, Pericardial effusion.
5, Diaphragm.
6, Oblique and transverse muscles.
7, Levator ani.
8, Muscles of the legs.
9, Femoral vein and valves.
10, Inferior vena cava.
11, Superior vena cava.
R.H., Right heart.
L.H., Left heart
A., Aorta.
P.V., Portal vein.

appear clear. Then the terrible dyspnœa of pericarditis with effusion occurred to me, and I perceived that this was the violent working of the respiratory force-pump, driving a gulp of blood from the abdomen through the right heart into the lungs at each inspiration and forcing it once more through the left heart into the aorta at each painful expiration. Allowing no sleep by night or day it was one long struggle of the skeletal muscles to maintain the circulation through the heart in spite of the strangling pressure of the pericardial effusion. It seemed, moreover, probable that this terrible dyspnœa worked in a pernicious cycle, and like the spreading œdema of the cerebral hemispheres tended to become progressively worse and worse. Each fluid wedge of blood driven through the compressed heart by the spasm of the abdominal muscles and the aspiration of the thorax acted across the incompressible effusion upon the softened pericardium, stretching it still more and making further room for fresh effusion. It then seemed clear that this type of dyspnœa was an imperative call for operative interference and the release of the effusion which was strangling the heart, for a pericardium so progressively dilated would probably not again contract so as to support the heart even were the effusion absorbed.

This matter seemed to be of such importance that I determined to investigate it experimentally, the research being carried out at the Brown Institution. A small rubber tube was ligatured into the pericardium of an animal under anæsthesia on Cohnheim's method, but in such a way that the thorax could be closed again and the animal resume normal respiration. Then warm oil was introduced into the pericardium, whilst a tracing of carotid and vena cava pressure was taken. The warm oil produced no effect whilst it filled the space in the pericardium which was not occupied by the heart, but directly it began to press upon the auricles the vena cava pressure rose and the arterial pressure fell and violent dyspnœa then followed, of the type which I had so hopelessly watched in several cases of pericarditis with effusion. Each gasping inspiration of the thorax was accompanied by violent contraction of the abdominal muscles and deep descent of the diaphragm, whilst expiration was accompanied by a sound which indicated obstruction of the larynx to the escape of air, thus raising the thoracic pressure and driving blood through the compressed left auricle and so into the aorta. That each respiration was

successful in forcing a gulp of blood from the veins to the lungs and from these to the aorta was shown by the wide oscillations of the carotid blood-pressure with each respiration. Directly the oil was allowed to escape from the pericardium the dyspnœa ceased, the carotid pressure rose, and the vena cava pressure fell to its usual low ebb, showing how beneficial the release of the fluid would be to patients suffering from pericarditis with effusion. I would therefore urge that dyspnœa of the type which I have described is the indication in pericarditis with effusion that the auricles are being pressed upon and that the effusion must be released. Of course this should be done by the open method and not by blind stabbing with a trocar and cannula, for there appears to be the greatest difficulty in distinguishing pericarditis with effusion from a widely dilated heart in a dilated pericardium. I know of a case where a young girl dropped back dead whilst the aspirating needle was being passed without an anæsthetic. A large pericardial effusion had been diagnosed, but on a *post-mortem* examination 15 ounces of pure blood were found in the pericardium and a wound in the heart. My experiments would seem, however, to indicate a diagnostic sign between these two conditions, although this has not been confirmed clinically. If tricuspid regurgitation is indicated by marked systolic pulsation in the veins of the neck the condition is probably one of a dilated heart in a dilated pericardium and not pericarditis with effusion. If the effusion exerts sufficient pressure to cause dyspnœa of the type which I have referred to, the heart will be compressed and the tricuspid valve will scarcely show relative incompetency. If the heart is so dilated as to simulate pericarditis with effusion the tricuspid valve must be widely patent during systole and the systolic pulse will be communicated to the veins. This brings me to another point. In animals relative incompetency of the tricuspid valve can only occur in a slight degree whilst the pericardium is intact. When the pericardium is removed and the heart is engorged with blood by pushing the chloroform or squeezing the abdomen the valves at once become incompetent and the systoles of the right ventricle appear as oscillations in the vena cava pressure. It would therefore appear to be a mistake to attribute relative incompetency of valves to stretching of their fibrous ring. It would seem to be due to dilatation of the pericardium.

We will now turn to some of the results which follow from the failure of the skeletal muscles to efficiently raise the blood from the legs and abdomen to the heart. When the muscles of the legs fail to act vigorously the venous blood stagnates and produces œdema and sweating of the feet, and in some cases varicose veins. It seems probable that loss of tone in the muscles of the calf is the cause of the association of varicose veins and sweating of the feet with flat foot. It is even more probable that varicose veins are produced by long-continued standing because the muscles do not periodically relax, allowing the deep veins to fill with blood and then pump it into the abdomen by a contraction. Pott's fracture, however, provides us with the best example of failure of the muscular venous pump in the leg. This fracture always involves the ankle-joint and is frequently followed by fibrous ankylosis. The muscles of the calf only serve to move the ankle-joint, so that when that joint is immovably fixed they cease to contract at regular intervals. As a result venous stagnation follows and the most obstinate œdema of the foot and leg, frequently going on to chronic ulcers. Ankylosis of the knee does not produce the same result, because the muscles of the thigh act also upon the hip-joint and continue to contract at intervals to move that joint. A similar œdema follows ankylosis of the wrist.

The moral to be drawn from the powerful vascular action of the skeletal muscles would appear to be that regular exercise to maintain the tone and power of the muscles is essential to the well-being of everyone, but more especially to people with a low blood-pressure. The development of the muscles of the abdominal wall is of the greatest importance, for a flaccid abdominal wall produces a languid venous return, and this again reacts upon the circulation of the brain and produces lassitude and depression. Especially will such general exercises as walking and cycling cause a rapid vascular flow all over the body and eliminate waste products from the tissues. The production of syncope by the relaxation of the arterioles of the abdominal organs is a well-known phenomenon. The blood leaks through into the capacious portal vein, and the ill-filled heart cannot supply the brain with blood. In fact, the man is bled into his abdominal veins. This could scarcely occur with a tonic and powerful abdominal wall, for a few deep breaths would rapidly return the venous blood to

the heart. It is apparently in anticipation of such emotional relaxation that Marryat makes his privateersman, and Kipling his soldier, take a reef in their belts or tightly tie a handkerchief round their waists before going into action.

But there is the commoner instance of what may be called partial syncope seen in nervous or overworked youths presenting themselves for examination. The diarrhœa and sickness point to abdominal congestion, and the mental confusion and loss of memory point to cerebral anæmia, whilst the thumping pulse and breathlessness indicate an ill-filled heart. Sometimes this condition is associated with albuminuria and forms one of the less recognised causes of the albuminuria of adolescence. The treatment of such people would appear to be obvious. There should be regular exercises, especially of the abdominal wall, and where such an ordeal as an examination has to be undergone an abdominal belt should be worn sufficiently tight to constrict the abdomen. I am convinced that neurotic and emotional people, who have, as a rule, a low blood-pressure, would escape from a large part of their depression and lassitude were they daily to perform calisthenic exercises and develop the tone of their abdominal wall. In this connection it is interesting to note the results obtained by Dr. Craig at Bethlem Hospital in cases of mania and of melancholy. He observed their blood-pressures, using for the purpose the sphygmometer invented by Dr. Leonard Hill and myself. In cases of mania associated with excessive restlessness he found the blood-pressure to be low, whilst in cases of melancholy with the sufferer sitting motionless in a stupor of grief he found the blood-pressure to be high. Further, he showed that in cases of mania the uniform pressure of a bath raised the blood-pressure and diminished the restlessness.

The PRESIDENT observed that when the author was describing the function of the pericardium, he himself was thinking of the network that surrounded the second bulb of a spray apparatus to prevent over-distension of the elastic reservoir. The author's remarks in reference to the condition in Pott's fracture called attention to the large amount of good that could be done by early and persistent massage.

Mr. WALLIS suggested that it was much more a medical than a surgical question, but there were one or two points brought out by the author which he would like to emphasise. One was as to varicose veins—so common a complaint in the out-patient departments of hospitals. He questioned whether the condition of the superficial veins quite corresponded to the facts to which the author had drawn attention. There

was no muscular action upon them as upon the deeper veins. The author's remarks in reference to pericarditis were of extreme interest from a surgical point of view. He had only once assisted at an operation upon the pericardium, and then it was by the open method; but unfortunately the patient died from the anæsthetic. He suggested that in addition to the open method they ought to make use of a local anæsthetic, such as eucaine, which was practically devoid of any toxic effects.

Mr. BARNARD, in reply, pointed out that the reason why the superficial veins became varicose was because the deeper veins were kept closed, owing to want of periodical contraction, in consequence whereof the blood had to pass through the superficial veins. He agreed with Mr. Wallis that it was a mistake to give a general anæsthetic to a patient suffering from pericarditis with effusion.

April 24th, 1899.

ON SOME COMPLICATED CASES OF APPENDICITIS AND THEIR SURGICAL TREATMENT.

By H. BETHAM ROBINSON, M.S., F.R.C.S.

To expound any new facts on such a well-debated subject as appendicitis might seem almost impossible, but the cases which I bring before you this evening, even if well recognised, will, I am sure, prove of interest, not only as illustrating some of the more serious complications of the disease, but as showing how the surgeon is required to be continually on the alert and what measures he may have to adopt to get a successful result. A group of cases I wish particularly to bring before you is where the abscess in relation with the appendix is attended by suppuration later in the neighbourhood of the liver, and we may first consider any factors that determine such a sequel. It would be difficult to show whether any particular position of the appendix is more liable to be attended by these complications, especially when we bear in mind how in many cases it is next to impossible to locate its exact direction. The most common site of the appendix is upwards and inwards towards the umbilicus, passing behind the inner margin of the cæcum and ileo-cæcal junction. It is conceivable that suppuration in relation with an appendix thus placed may have more chance of becoming encysted through the early matting together of the folds bounding the ileo-cæcal fossa. If the base of the appendix be the situation of the lesion

or the appendix be placed in the iliac fossa or running up on the outer side of the cæcum, the encysting of the pus is much more dependent on peritoneal adhesions and matted coils of bowel, as the parieto-colic folds with their ordinary development would not have much limiting influence. It is my experience that the adhesions usually are weaker towards the outer side in the direction of the ascending colon, and acting on this it is my special care to very gently handle the parts in this direction. By recalling the anatomy of this region one can imagine (if there are no restraining adhesions and the peritoneum gets fouled on the outer side of the cæcum) how very easy it is for the infection to spread upwards. The recumbent position and the inclined plane on the outer side of the ascending colon leading to the space over the head of the kidney are factors of importance in the accumulation of pus beneath the liver. This intraperitoneal space in the right hypochondrium can contain a large fluid collection, and is very readily shut off along its inner side from the general peritoneal cavity. In front are the under surface of the right lobe of the liver and the gall-bladder, both of which are in relation below with the hepatic flexure of the colon; above is the reflection of the peritoneum from the liver and right lateral ligament to the parietal peritoneum over the right kidney and posterior abdominal wall to its outer side; on its inner side are the foramen of Winslow and duodenum; and below is the hepatic flexure of the colon with the commencement of the space along the outer side of the ascending colon. Rutherford Morison* has drawn attention to this space as a seat for fluid collections after gall-bladder operations. It would be expected that a collection here might drain into the lesser sac of the peritoneum, but no doubt this is early sealed off by adhesions, and I have determined that this was so in one of my cases. It also must be borne in mind that no mechanical overflow could take place until late owing to the depth of the fossa posteriorly below the level of the spine, the foramen of Winslow being of course anterior to the spine. The most likely extension from this space would be over the anterior and superior surfaces of the right lobe of the liver round the inferior margin of the gland or round the edge of the right lateral ligament, forming a subdiaphragmatic collection.

* 'British Medical Journal,' vol. ii, 1894, p. 968.

With these preliminary anatomical observations I will quote cases which I think illustrate these points.

Case 1. *Gangrene of the Appendix with Abscess; Secondary Collection below the Right Lobe of the Liver; Operations; Recovery.*—A youth, aged 18 years, was admitted into St. Thomas's Hospital on January 21st, 1898, under the care of my colleague, Dr. Payne. Having previously always been well, the patient had abdominal discomfort on the 19th with loss of appetite, culminating in pain generally distributed at night without nausea. There was little sleep that night but the next morning he seemed better, ate some breakfast, and went to his work; he also took some salts. During the day there were nausea, a good deal of hiccough, and loss of appetite; in the afternoon and night the pain seemed to localise itself to the right iliac fossa, causing considerable tenderness. On admission he had a flushed face and furred tongue with a temperature of 102° F. The abdomen was hard and tense on the right side, especially at the lower part over the iliac fossa where there was considerable tenderness with dulness on percussion. Only the upper part of the abdomen moved during respiration and then only to a slight extent. The abdominal muscles on the right side were contracted so that the last rib and iliac crest were approximated; on palpation a well-marked mass could be detected with special tenderness over McBurney's point. The patient said that on arriving at the hospital he noticed the pain, which had been before in the iliac fossa, shoot up his right side; this lasted for some time and then left an aching pain, which soon, however, disappeared. The liver dulness was normal and there was nothing to be detected in the chest. On the 22nd the temperature was 100·4°, but the pain was less; there had been constipation for three days. In the afternoon an operation was performed and a collection of pus was found behind the cæcum surrounding the appendix which was directed upwards towards the umbilicus. The appendix was so firmly adherent at its distal end as not to permit of its separation with safety; its central part, which could be easily freed, was gangrenous on one side. As the whole appendix could not be removed the gangrenous part was incised and a small drainage tube was inserted and lightly packed round with gauze. The temperature after the operation dropped to normal, but after a few hours began to rise again and he was troubled with slight cough. On the morning of the 24th the temperature was 101·4°, the respirations were 34, and the pulse was 127. The cough was still troublesome and there was some purulent expectoration. The right side of the chest was dull in front from the fourth rib downwards and from the inferior angle of the scapula behind; over this dull area the breath sounds were diminished and the voice sounds were ægophonic. He was again examined and it was found that a track ran upwards from the wound to the dull area along the ascending colon. An incision was then made just below the costal margin on the right side about 4 inches long dividing the outer part of the rectus. Thin, purulent, offensive fluid was found occupying a space bounded by the inferior surface of the liver, the front of the kidney, the hepatic flexure of the colon and the duodenum and matted gastro-hepatic omentum on the left. Into this space the probe could be passed up along the ascending colon. There was some fibrinous deposit about the inferior border of the liver creeping round on to the anterior surface. Two tubes were then inserted, one

draining the space through the loin and the other tube passing down in the line of the colon to the lower wound. The next morning (the 25th) the temperature was 99°, the pulse was 102, and the respirations were 32. There was no pain except on coughing and then only in the wound. Through the day and night the temperature rose again, reaching 101·4° at 8 a.m. on the 26th. His general condition was extremely precarious as he was cyanosed, with bubbling crepitations over both lungs, and the discharge from the wounds was extremely offensive. Fortunately he rallied, the temperature going down during the day, reaching 98° at midnight and then remained below 100°. The lungs gradually cleared of moist sounds but on the 28th there was some impairment of the percussion note with slight friction in the left post-axillary line at the level of the 11th and 12th dorsal spines. These disappeared during the next day or two and his further progress was almost uneventful except for a little anxiety owing to the temperature going up on February 7th and remaining so until the 13th, when it finally became normal. This rise was associated with signs of consolidation at the right base. Both drainage tubes were removed on the 11th, but the wounds were a long time closing, the top one being healed on March 17th and the lower one shortly afterwards.

Case 2. *Appendicitis with Abscess; Secondary Collection below the Right Lobe of the Liver; Operations; Recovery.*—A child, aged 8 years, was admitted into the East London Hospital for Children, Shadwell, under the care of Dr. Donkin, on August 5th, 1898. There was no history of any previous attack. Four days before admission the illness started with vomiting, drowsiness, and fever, and the next day pain was complained of in the right iliac region. All the next day she had the pain and at night the mother gave her castor oil, when she vomited for the second time. On the afternoon of the fourth day she was admitted into the hospital. The bowels had not acted since the commencement of the illness and nothing but milk had been taken. On admission the abdomen hardly moved during respiration and the right hip was flexed. There was very great tenderness, and a lump could be felt in the right iliac fossa. Per rectum no lump could be felt bulging from the right side. The temperature was 101° F. and the pulse was 108. At midnight the temperature was 102°, but during the 6th it dropped to 99·4°, rising at night to 100·8°. On the 7th the patient seemed decidedly better, the temperature being lower, but on the 8th after rising to 100·6° it fell to normal at midday, only to rise again to its original level. The dull area had extended more into the loin and indefinite fluctuation was obtained. On the 9th, the temperature having continued to rise to 102°, I was asked to see her, and as there was undoubtedly pus present immediate operation was advised. The usual skin incision was made and the layers of the abdominal wall were split. On incising the peritoneum a pus cavity was opened which extended up behind the inner side of the cæcum and ileo-cæcal valve in the line of the normal direction of the appendix. The appendix was embedded in adhesions and no lesion was found in it. The cavity was firmly walled in by adhesions below, but on the outer aspect of the cæcum they were very delicate. The pus was most offensive. A rubber tube was inserted into the cavity and iodoform gauze was lightly packed around it. The temperature was 96° after the operation and rose again to 100°, gradually coming to normal during the next 24 hours. At midday on the 14th after everything had proceeded satis-

factorily the temperature went up to 101° and remained so with only slight remission up to the afternoon of the 16th, when the second exploration was made. On examination on the 16th there was well-defined fulness on the right side of the abdomen below the costal arch and also in the right loin. There was noticed approximation of the last rib and the iliac crest. With respiration there was very little movement on the right side. Dulness extended up from the wound to the costal margin and into the right loin, and a fluid thrill could be elicited from front to back. The upper margin of the liver dulness was in the fifth space in the nipple line. From the first wound the discharge was now much thinner and less offensive. As it seemed clear that there was a second collection of pus an incision was made just below and parallel to the costal margin from the linea semilunaris into the right loin. An intraperitoneal pus collection of almost half a pint was opened. This space had for its boundaries the under surface of the liver above, the foramen of Winslow and the duodenum on the inner side, the hepatic flexure of the colon below, and it was completely shut off from the previous collection. There was no extension in front over the anterior surface of the liver. The cavity was washed out with boric lotion and a gauze drain was inserted. The after-effect on the temperature was excellent, as it was normal except for a very temporary rise to 100° on the 21st. On the 19th she had jaundice, which perhaps was not surprising considering that the common duct was in the inner wall of the cavity, but this soon disappeared. There is nothing to detail about the further progress of the case. The lower wound was healed at the end of the month but the upper one was not sound until the middle of September, when she went away to the convalescent home.

The above two cases are very much of the same type, each of them having, secondary to the primary lesion, a well-defined collection under the right lobe of the liver and limited to that region. In Case 1 the physical signs in both lungs after the operation did not appear to have any relation to the inflammation about the liver. From the extension of the liver dulness upwards it was a question before the secondary operation whether the organ was simply pushed up or whether there was as well pleural effusion on this side or a subdiaphragmatic collection. After the operation this upward extension soon disappeared, so it seemed proved that the organ had only been displaced upwards.

Case 3. *Appendicitis with Abscess in the Pelvis; Secondary Inflammation about the Liver; Subdiaphragmatic Abscess (?); Right Empyema; Parotid Bubo; Operations; Recovery.*—A boy, aged 16 years, was admitted into St. Thomas's Hospital under the care of Dr. Payne on March 15th, 1898. Up to the present illness he had always enjoyed good health. About four weeks before admission he complained of abdominal pain; a fortnight later he had severe vomiting and took to his bed. He was admitted into the hospital with a rigid abdomen, more especially in its lower part, the condition simulating an over-distended bladder. On drawing off the urine no difference was produced in the

size of the swelling. Rectal examination proved the existence of fluid in Douglas's pouch. The temperature was 102° F., the pulse was 100, and the respirations were 28. In the evening his abdomen was explored by a lateral incision as his symptoms pointed to appendix trouble. The muscular layers were split, not cut, and the peritoneum was opened. Here it was found that matted bowel roofed in the fluid collection, and to get at the latter the coils were gently separated and about one pint of pus with some fæcal matter was set free. The appendix was not found. A glass tube was put into the pelvis and a gauze drain into the upper part of the wound. His progress for a week was fairly satisfactory, and then his temperature began to go up, reaching from 102° to 103°. On March 22nd two small rubber tubes were substituted for the previous drains, as the discharge had considerably lessened. On the 23rd he was noticed to be developing a parotid bubo on the left side, which remained for some time without showing any signs of softening, but eventually an abscess formed and it was opened on April 6th. A large piece of necrosed tissue, apparently lymph, came away on the 2nd from the abdominal wound followed by a considerable quantity of foul-smelling pus. There was marked diminution in the extent of the dull area and of abdominal tenderness. On the 7th he had severe pain on the left side below the cardiac area, and friction could be heard along the line of the seventh rib into the axilla. There was some dulness behind from the angle of the scapula downwards. On the 8th he complained of pain in the abdomen, and on coughing brought up several ounces of thin muco-purulent sputum with very offensive odour. On examination on the 9th the abdomen was not painful or tender and moved fairly well and equally on respiration. The impaired resonance remained the same on the left side without evidence of fluid collecting. The parotid bubo had discharged freely and the temperature had gone down. There was complaint of pain in the right shoulder. The temperature came down to normal on the 11th, but the lung-signs on the left side remained ; if anything, the breath-sounds were less marked. On the 13th the temperature went up again to 102·8°, and the next morning there were signs of fluid in the right pleura with friction in the axilla and diminished breath and voice sounds at the base in front and behind. The liver dulness began at the fifth rib in the nipple line, and at the sixth in the mid-axilla. The upper part of the abdomen was explored on the 16th by an incision parallel with the right costal margin starting from the linea semilunaris. Dense adhesions were found on the upper and under surfaces of the liver, but no pus was discovered. A hypodermic needle was passed into the pleura and a negative result was obtained. On the 27th the physical signs at both bases continued as before, except that now there was no friction on the right side. He was cheerful, had no pain, slept well, and took his food readily, but the temperature was still irregular. On May 6th there was some return of abdominal pain, but no lesion was detected to explain this. The parotid wound had almost healed. The upper abdominal wound had closed, but the lower one still discharged. On the 16th the physical signs in the lungs were the same, but the persistence of the nightly rise of temperature strongly suggested pus. It was determined to explore the right chest again on the 25th, as some œdema was noticed over the lower spaces laterally. An incision was made over the right tenth rib in the posterior part of the axillary line and about 2 inches of bone were removed. On opening the pleura very foul pus was evacuated from a localised cavity

apparently pleural ; this was washed out with boric acid, and as there was considerable hæmorrhage the space had to be plugged with cyanide gauze for some hours. After the operation the temperature fell to 96·4° and then rose again to just over the normal line. At this period the patient was in a terribly emaciated state, weighing only 4 st. 6 lbs. From this time on he rapidly improved, his temperature remaining normal excepting for a sudden unexplained rise to 103·6° on June 3rd. He took his food well and gained flesh, reaching 5 st. at the end of the month, by which time his wound had healed. He was discharged from the hospital on July 10th.

The appendix abscess in this case appears to have gradually collected in the pelvis and to have been roofed in by matted small intestine. As to the position of the appendix I cannot say for certain, but there is a strong probability that its direction was downwards and inwards towards the pelvis and possibly overhanging the pelvic brim. As in the former cases, there was spreading of peritoneal infection along the colon to the liver as was shown by the extensive adhesions on exploration. There was no actual proof of any subdiaphragmatic collection, but the lung signs terminating in the basal empyema on the right side gave some colouring to this belief. The association of the parotid bubo may be noted as it has been so frequently in relation with peritoneal inflammation especially when in the pelvis.

The next case is one of a different type and of considerable interest from the subacute nature of the symptoms and from the local conditions found on exploration.

Case 4. *Appendicitis ; Secondary Abscess below the Costal Margin superficially placed ; Fæcal Fistula ; Recovery.*—A boy, aged 12 years, was admitted into St. Thomas's Hospital under the care of my colleague, Dr. Payne, on April 13th, 1898. He had always had good health, and so this seemed undoubtedly his first attack. On April 7th he was seized with severe pain in the abdomen, and the next day he commenced to vomit, which continued up to the date af admission. During these days practically no nourishment was taken. The bowels were last opened on April 6th. On admission he was found to be well nourished but pale. The abdomen was rather distended, and did not move well on respiration. It was resonant all over except in the right iliac fossa, where a distinct tumour could be felt. Over this area it was extremely tender, especially over McBurney's point. The rest of the abdomen was not tender on palpation, the liver dulness was normal, and the spleen was not felt. His temperature was normal, and the pulse was 90. During the next few days there was gradual loss of tenderness with reduction in size of the inflammatory mass. He vomited once or twice, but was able to take fluid nourishment. His bowels were opened naturally on the 18th. His temperature reached 100° F. on the three successive evenings of the 14th, 15th, and 16th, but was otherwise normal. On the evening

of the 22nd, after having a more substantial diet, he had a severe attack of vomiting. On examination, a hard lump of about the size of a walnut could be felt at the outer edge of the right rectus, about 1 inch below the costal margin; this seemed in the abdominal wall; it did not move with respiration, and was tender on pressure. Nothing could now be felt in the iliac fossa, but there was a cord-like induration starting at about the lower quarter of the rectus, running upwards along its outer margin to the lump above. On the 25th I operated, making an incision over the upper lump parallel to the costal margin; beneath the transversalis a little pocket of very fœtid pus was opened. An incision was then made below over McBurney's point, and the lower end of the cord-like thickening was exposed, and again a little pus was set free. A probe was passed into the lower end of what proved to be a sinus lined with soft granulations which led up to the small abscess. Downwards the probe went towards the pelvis, and this track was opened up by dividing some of the outer fibres of the rectus and the deep epigastric artery. No more pus was discovered. The probe could be easily felt in the pelvis per rectum in relation with its right wall, but it was difficult from this site and from what was found in the wound to say whether the granulation track was in the subperitoneal layer or among peritoneal adhesions. No bowel was displayed. A drainage tube was put in the lower track, and a gauze drain in the upper one; the wound was partially sutured. His condition after this for some days was satisfactory, but then his temperature began to go up at night, reaching its maximum (102·2°) on the 7th, on which day there was a fæcal discharge from the wound, and after that a gradual reduction in temperature. On the 14th the lower sinus was again explored, when at the bottom an opening was found in the bowel admitting the tip of the finger; all round the opening was granulation tissue, and it was difficult to say whether the bowel so perforated was the base of a dilated appendix or the cæcum, but I thought the latter. From the rotten state of the bowel wall it was impossible to close the opening without doing an extensive operation, and so it was not attempted. His upper wounds soon healed, and the fæcal fistula gradually contracted but yet allowed considerable discharge when he went to the convalescent home on July 1st. He came into the hospital again in December, his fistula having contracted up to a very small opening; as there was only a slight fæcal discharge which could be easily controlled, I determined to wait events, knowing how unsatisfactory attempts to close these fistulæ are. Since then I have seen nothing more of him.

The main feature of interest in this case was the possible unusual sequence of events after the primary lesion. On exploration it was extremely difficult to say whether the pus collection outside the margin of the rectus was in the subperitoneal tissue or in the peritoneal cavity. If in the subperitoneal tissue it must be assumed that the peritoneum had become adherent about the base of the appendix or cæcum and then given way, leading to the gradual travelling up of the pus towards the costal margin. If the lesion was intraperitoneal we have the same course of events as in the previous cases, only on a smaller and less active

scale. The infection spread up on the anterior surface or outer margin of the colon, the latter becoming adherent to the abdominal wall. Now an apparent difficulty, the abscess just below the costal margin, is not so, for if the lower edge of the liver were adherent to the colon and both of them to the parietal peritoneum at the costal margin the pus might easily make its way forwards. The ease with which the probe in the bottom of the wound could be felt per rectum also gives some support to the intraperitoneal site, for it would pass among adhesions into Douglas's pouch, not an uncommon direction for pus to take, whereas if this track had been in the subperitoneal tissue the probe would have seemed more distant.

To summarise, the following points may be deduced from this paper:—(1) In a small proportion of cases of appendicitis with abscess, there is intraperitoneal extension to the liver region; (2) in relation with the liver, pus may be only below it or also above it "subdiaphragmatic"; and (3) clinically there is considerable difficulty in determining when the apparent upper border of the liver is raised, how much of this is due to simple displacement upwards by a fluid collection or to a subdiaphragmatic abscess or to an associated lesion at the lung bases.

Mr. Battle congratulated Mr. Robinson upon being able to show such a brilliant series of cases, and observed that only those who had to treat them knew the labour and anxiety caused by such complications. It was bad enough to have to make the diagnosis in the first place, and to get a patient started on the road to recovery; but when this was retarded by such a series of complications, it really looked as if the fates were banded against them. He himself had only been called upon to treat subdiaphragmatic abscess dependent upon peritonitis on three occasions, and only one of them was at all similar to the first case related by the author. It was that of a boy admitted to hospital in September, last year, with symptoms of four days' duration pointing to acute septic peritonitis. He made an incision over the appendix, and removed the gangrenous appendix. There was much offensive pus in the peritoneum, but he did not care to disturb the adhesions to ascertain its limits. Some time later, he opened a huge abscess extending to the upper surface of the liver. This afterwards made its way through the diaphragm into the pleura, and a definite hole could be felt in the diaphragm. The patient recovered, but only after rather a severe operation, involving a large incision and resection of portions of the ribs. He thought there was little doubt that these collections tracked up along the peritoneal surface of the bowel, but not in the cellular tissue, and had collected immediately beneath the diaphragm, on one side or the other, as the case might be. In one case, in which the diagnosis had been made of subdiaphragmatic abscess secondary to peritonitis, the pus was thought to be between the spleen and the stomach above the transverse colon. He made an incision through the

diaphragm, in the splenic region, but the spleen was so much enlarged and so firmly adherent that he was unable to pass in any instrument to reach the collection. He therefore made an incision in the left linea semilunaris, but could find nothing. In another case of the kind he explored the region in which the abscess was supposed to be, but it was not a large one, and he was unable to feel it, in fact it was only discovered *post-mortem*.

Dr. Hadley observed that these cases were certainly very interesting to the surgeon, but they might also be of importance to the physician. He had, with his surgical colleague, seen a case something like those related by the author. The patient was admitted with symptoms of localised peritonitis in the upper part of the abdomen, but without any symptoms at all referable to the cæcum. On operation a little pus was evacuated from the neighbourhood of the liver. The patient, however, did not do well, and the temperature kept up. He was then asked to examine the right pleura. He diagnosed fluid, and a little clear fluid was evacuated, but it was a curious feature that the heart was greatly displaced. To judge from the symptoms in the right pleura, one would have imagined that there was a good deal of fluid there. The dulness did not much diminish, but the heart went back to its place. About three weeks afterwards he saw the patient again, when he found that the lung was practically clear, but there was still the discharging sinus below the liver, and a further exploratory operation was suggested. This was done, with the result that it was found that the whole of the trouble beneath the liver, reaching back deeply towards the spine, had started from an abscess round the cæcum.

Mr. McAdam Eccles thought the author had done good service in emphasising the fact that many of these remote collections were intra-peritoneal, and not subserous. He referred to a case under his care three years ago, of a boy with obvious signs of peritonitis in the right iliac fossa, not, however, definitely pointing to the appendix. He saw him six days after the onset of the attack, when there was a collection of pus which could easily be felt per rectum. This was evacuated in the right iliac fossa, though not so thoroughly as might have been wished. Some time later the patient's temperature again rose, and a collection of fluid became obvious on palpation and percussion in the right loin. He made an incision in the lumbar region and found pus, distinctly intra-peritoneal, on the outer side of the ascending colon, about the region of the lower end of the kidney. He suggested that in many of these cases the tracking was due to the want of a free incision, and he thought it might sometimes be possible to prevent its occurrence by making a counter-incision in the loin at the same time as the primary incision in front. He asked whether the author had been enabled to form any opinion as to the value of a counter-incision in such cases.

Mr. Betham Robinson, in reply, admitted that if one knew that there was likely to be such a collection, a counter-incision would be a grand thing, but one saw so many cases in which it was not necessary that it would be doing too much to make a loin incision right away as a routine measure.

THE RELATIVE EXCRETION OF THE NITROGENOUS WASTE PRODUCTS IN A CASE OF LIVER ABSCESS.

By William Bain, M.D., M.R.C.P.

Our knowledge regarding the influence of the liver on the intermediary products of proteid metabolism has been to some extent derived from analysis of the urine, therefore it is important in hepatic disease to make quantitative estimations of urinary constituents likely to be affected either absolutely or relatively by impairment of its functions. Results indicating abnormal metabolism have been obtained in certain diseases of the liver. For example, in cirrhosis the preformed ammonia is increased at the expense of the urea, while in acute yellow atrophy a similar change occurs, viz., a considerable increase in the former associated with the almost total disappearance of the latter. Until recently it was generally held that the urea was markedly diminished in phosphorus poisoning, but Munzer's researches * negative this view. Indeed, there is an increase in the urea relative to the nitrogen excretion resulting from tissue disintegration. The uric acid and phosphoric acid are greatly increased, the increase in the former corresponding to, and in the latter being relative to, the increased nitrogen excretion. The preformed ammonia is also augmented, due probably to a diminished alkalinity of the blood. Lactic acid is frequently present. Although one scarcely expected to find any very marked deviation from the normal in a case where only a portion of the liver was affected, still it was considered probable that an examination of the urine might reveal some slight modification in the relative excretion of the nitrogenous waste products. For permission to examine and report the following case I am indebted to Mr. Makins, surgeon to St. Thomas's Hospital :—

C. P., aged 31, was admitted into St. Thomas's Hospital on November 17th, and died on December 23rd, 1898.

History of the Case.—Last July he had an attack of dysentery when in China, which lasted a couple of weeks. Three weeks later the attack recurred and persisted for about a month, being accompanied by jaundice. On the voyage home he began to suffer from pain in the hepatic region and under the right shoulder.

* Munzer, 'D'Archif. f. Klin. Med.,' xii, 1894.

On admission, the liver dulness was found to begin above at the fifth rib in the nipple line, and extended downwards to the umbilicus. He was operated upon the same day, the liver being exposed, but not explored. On November 21st a trocar and cannula was inserted into the liver, when about 48 ounces of chocolate-coloured pus welled up. The wound continued to discharge large quantities of fluid daily. The sediment from this fluid was examined by Mr. Shattock, who found it to consist of necrosed liver cells. There was a moderate degree of pyrexia, and the patient became progressively weaker and gradually sank.

Post-mortem Appearances.—On opening the abdomen there was evidence of peritonitis beneath the under surface of the liver, chiefly in the region of the right lobe and the head of the right kidney. The liver was greatly enlarged both upwards and downwards, its anterior surface in the upper part being firmly adherent to and incorporated with the diaphragm. Incision showed that the right lobe of the liver was the seat of some six abscess-like areas, mostly still solid, but commencing to soften in places. The average size of these areas was that of an orange, their form rounded, whitish or greyish in colour, and they were distinctly marked off from the surrounding liver substance. When the softening was in the early stage their substance was channelled by small excavations, giving them a honeycombed appearance. In others its advance was indicated by the welling up of thick greenish pus, and it was one of these that had been drained by operation. The left lobe of the liver was entirely free from such areas, but contained a few firm white nodules, in diameter about the size of an ordinary lead pencil. The substance of the right lobe was obviously diseased, being of a mottled red and white colour. In both kidneys the cortex was increased and also abnormal in appearance, being much streaked and congested. There was an ulcer in the small intestine, and another at the upper end of the rectum. These penetrated to the muscular coat, which was covered with pus, and their edges were thinned and raised. No other change was apparent in the mucous membrane. The remaining organs were normal, and no further evidence of septicæmia was observed.

The analysis of the urine included the determination of the total daily excretion of urea, uric acid, phosphoric acid, ammonia, and alloxur bases. Lactic acid was estimated on one occasion.

Methods of Analysis.—For the estimation of urea the hypobromite method was employed, the solution being made afresh each day. Hopkins's method was adopted for estimating uric acid; Schlösing's method for ammonia, and phosphoric acid by titration with uranium nitrate.

In estimating lactic acid, 1 c.c. of a 25 per cent. solution of sulphuric acid was added to 100 c.c. of urine, and then barium hydrate until no more precipitate appeared. The baryta was precipitated by passing a stream of carbonic acid gas through the solution, which was then filtered, and the filtrate evaporated to a thin syrup over a water bath, the temperature of the bath not exceeding 70° C. About 10 volumes of alcohol were gradually added to the syrupy fluid and then evaporated. This was repeated, and subsequently a few drops of dilute phosphoric acid added to the residue, which was then extracted with an excess of ether. The ethereal solution being separated and carefully evaporated the solid material which remained consisted entirely of lactic acid. This was dried and weighed.

To determine the alloxur bases Salkowski's recent method was adopted;

500 c.c. of urine were taken the first five days but only 150 c.c. the remaining two days, as the quantity excreted was small. The phosphates were precipitated by ammonia and magnesia mixture, and removed by filtration. Three per cent. silver nitrate solution (6 c.c. to 100 c.c. of the filtrate) was then added, and after brisk stirring the whole set aside for an hour or more in a dark place. Some of the supernatant fluid having been examined to see if there were a sufficiency of silver, the solution was filtered and the precipitate washed chlorine and silver free. The precipitate was now decomposed by passing H_2S through it in acid solution. The fluid was then heated, filtered, and evaporated to dryness on a water bath. The residue was treated with about 30 c.c. dilute sulphuric acid, brought to the boiling point and left in a cool place for 20 hours. The precipitate (uric acid) was then collected and washed with dilute sulphuric acid. The filtrate and washings, amounting to about 50 c.c., were rendered ammoniacal, silver nitrate solution added, the precipitate so obtained was filtered, washed chlorine silver and sulphate free, and then incinerated in a porcelain capsule. The ash was extracted with dilute nitric acid and the silver estimated by titration with NH_4CNS. From this silver determination the amount of alloxur base is calculated from the average amount of silver contained in the silver compounds of the different bases.

As it was impossible to put the patient on a fixed diet while the urine was being analysed, the diet chart is here inserted.

Diet in the 24 Hours.

Dec. 13th.—No record.

„ 14th.—6 pints of milk, 1 egg, 1 sponge, leg of pheasant, 6 pieces of thin bread and butter, 1 round dry bread, and 6 ounces brandy.

„ 15th.—5½ pints of milk, 3 pieces dry bread, 3 pieces thin bread and butter, leg of rabbit, plate of pudding, 1 egg, and 4 plain biscuits.

„ 16th.—3½ pints of milk, 2 pieces of bread, 1 round of bread and butter, 1 plate of mutton, 1 plate of milk pudding, and 6 ounces of brandy.

„ 17th.—3½ pints of milk, 2 pieces bread and butter, 1 egg, wing of chicken, 1 piece dry bread, 1 pint of arrowroot with egg beaten up, and 6 ounces brandy.

„ 19th.—4 pints of milk, 1 egg, custard pudding, 1 round bread and butter, half slice of bread, and 6 ounces brandy.

„ 20th.—½ pint of milk, 1 egg, 2 pints of tea, ½ pint coffee, 2 basins arrowroot, 1 piece of toast, and 6 ounces brandy.

Table showing the Quantitative Estimations of the more important Constituents of the Urine in the 24 *hours.*

Date.	Quantity, in c.c.	Urea, in grms.	Uric acid, in grms.	P_2O_5, in grms.	Ammonia, in grms.	Alloxur bases, in grms.	Lactic acid, in grms.
Dec. 13th ..	778	22·414	·787	1·07	·661	·0739	—
„ 14th ..	820	14·824	·529	1·06	·721	—	—
„ 15th ..	762	21·008	·702	·61	·583	·0699	—
„ 16th ..	1,050	20·180	·704	·85	·848	·0668	—
„ 17th ..	1,088	21·876	·622	·95	·925	·0604	—
„ 19th ..	460	12·344	·601	·59	—	·0872	—
„ 20th ..	400	13·276	·537	·68	·450	·0631	·360
Averages ..	765	17·974	·64	·83	·69	·069	·36

Taking into consideration the diet and excluding lactic acid, the results obtained must be regarded as coming within the normal physiological limits. Notwithstanding the negative character of these results they are interesting, inasmuch as they show us that although portions of the liver were distinctly diseased, yet its functions were apparently efficiently performed. Regarding urea and ammonia no comment is necessary.

The phosphates are undoubtedly diminished, but this diminution is evidently due to the character of the food taken. The greater part of the phosphoric acid in the urine is directly derived from the ingesta, but a small portion is a decomposition product of phosphorised organic material, such as nuclein. If the same amount of phosphates were taken in the food each day the variations occurring in the excretion of phosphoric acid would be a measure of the amount of disintegration of nuclein containing tissues.

Sarcolactic acid is not a constituent of normal human urine. It was only estimated once in this case, when the patient was in a

semi-moribund condition, and as it has been observed in many diseases shortly before death, no importance can here be attached to its presence.

The interest in uric acid arises chiefly from the influence it is supposed to exercise in the causation of gout. The amount excreted in health varies considerably in different individuals, and this variation by no means necessarily depends upon the food taken. If a healthy subject is placed upon a fixed diet, other conditions remaining as much as possible the same, the excretion still shows daily variations. If the uric acid were derived entirely from the ingesta, the only possible explanation of this variation would be that although the food taken was the same in kind and quantity, the amount assimilated varied, but as this is more or less of an assumption, there is another supposed origin of this acid which will be considered. It is well known that nuclein and nucleo-proteid greatly increase the excretion, and Horbaczewski's view that uric acid is derived from the nuclein of the white cells imparts additional interest to the question of the mode of production of this acid. Some support has been given to this theory by experiments recently recorded in the 'Journal of Physiology,'* in which it has been shown that the variations in the number of leucocytes have an inverse ratio to the variations in the excretion of uric acid, but it is difficult to understand why this nuclear origin should be restricted to the leucocytes. If uric acid is formed from disintegration of leucocytes, in all probability the other tissue cells share in its production. If at the same time the phosphoric acid were shown to follow closely the uric acid excretion, the evidence in favour of the nuclear origin would be strengthened.

The relationship of the alloxur bases to uric acid is a very close one. They are built upon the same alloxan nucleus, they are produced on the decomposition of nuclein, and Horbaczewski† showed that spleen pulp was able to form uric acid from xanthin bases. The amount of the alloxur bases excreted in the 24 hours is roughly about one-tenth of the uric acid, and they vary directly with the acid.

* Bain and Edgecombe, 'Journal of Physiology,' 1899.

† Horbaczewski, 'Sitzungsbericht der K. Acad. d. Wiss in Wien,' ciii, 1891.

THE INFLUENCE OF NITROGEN GAS ON THE EXCRETION OF URIC ACID.

By Wm. Armstrong, M.R.C.S. (Buxton).

The Goulstonian Lectures of 1897, in which Dr. Luff so ably dealt with "The Chemistry and Pathology of Gout," marked a distinct advance in the treatment of that disease. It was shown that in the Gowland Hopkins method there was at command a test which, while it showed the exact amount of uric acid passed, did not throw down the xanthins and hyper-xanthins—the great blot on certain other methods in general use; the necessity for the purpose of exact observation of testing a sample of the mixed urine of the 24 hours was pointed out; and, more than all, it was demonstrated clearly and accurately that the gouty patient, so far from passing uric acid in excess, in the great majority of instances passed too little, and that there was a daily storage going on which, although small in amount, was still sufficient to keep up certain morbid conditions; or if it had no such gradual outlet eventually culminated in an acute attack of articular gout. In repeating these experiments I observed that in patients drinking or bathing in the Buxton thermal water there was a constant, and in some cases a considerable, increase in the quantity of uric acid excreted, and that this action was the more marked as the natural temperature (82° F.) was approached. Neither the mineral constituents nor the temperature of the water accounted for the facts observed, while nitrogen gas, although present in very large quantity, had always been looked upon as a simple element which was therapeutically inert. It seemed rather significant, however, that Wilbad, Gastein, and Buxton—all associated with relief to the gouty—possessed nothing in common, both temperature and mineral constituents differing—except the presence of nitrogen, and it could not be overlooked that the recent researches of Lord Rayleigh and Professor Ramsey had shown that gas to be not an element but really a very complex body, the whole chemistry of which is probably not yet known.

With a view to finding, if possible, the *rationale* of the "gout-cure" for which the Buxton thermal water had so long been

noted, a large number of observations and many central and cross experiments have been made. Over three hundred persons have contributed to this investigation. They were of various ages; all except 10 suffered from gout in one form or another; these 10 were persons in normal health showing no sign of the gouty diathesis. Their ordinary mixed dietary was not altered in any way. Some of the cases only drank the thermal water, 24 ounces being taken daily, divided into three doses; some bathed only, while a third section both drank and bathed. In all cases the 24 hours' urine was tested, and the first two examinations were made before either the bathing or drinking cures were commenced.

The Buxton thermal water is discharged from the earth at a temperature of 82° F. It is soft, clear, and of a light bluish colour, almost free from taste and without odour. Several exhaustive analyses have been made, the most complete by Dr. Thresh, who devoted much time and care to the investigation.

His results were as follow:—

Calculated into grains per gallon, the water was estimated to contain—

Bicarbonate of calcium	14·01
,, magnesium	6·02
,, iron	·03
,, manganese	·03
Sulphate of barium	·05
,, calcium	·26
,, potassium	·62
,, sodium	·84
Nitrate of sodium	·03
Chloride of calcium	·02
,, sodium	3·10
,, ammonium	Trace
,, magnesium	·95
Silicic acid	·95
Organic matter	·02
Carbon dioxide	·20
Nitrogen	·19
	27·32

Lithium, strontium, lead, and phosphoric acid traces.

There were 6 cubic inches of nitrogen to the gallon and a small quantity of carbonic acid gas. This nitrogen has been carefully tested and contains 2 per cent. of argon and a trace of helium.

It will be seen that the mineral constituents are small in quantity and of little therapeutic power, and that there is a large quantity of nitrogen gas, a little in suspension, but by far the greater quantity in solution.

The following lines were laid down for the purpose of this investigation :—

(1) To note the effect of drinking the thermal water as it emerged from the spring.
(2) To eliminate (*a*) by cooling, (*b*) by heating the contained gases and note the comparative effects.
(3) To test the relative effects of drinking water at the same temperature.
(4) To note the effects of bathing in the thermal water at 82° F., 90° F., 96° F., 100° F., and in ordinary water at 82° F., and at 100° F.

The average quantity of urine for the 24 hours before treatment commenced was a little under 46 ounces in the non-gouty and 41 ounces in the gouty ; in the former the uric acid averaged ·780 grammes per day, in the latter ·660 grammes.

Drinking. (Over 60 cases.)

(1) Thermal water drank at spring at 82° F.—Average urine after one week, 57 ounces ; uric acid, ·799 grammes.
(2) Thermal water allowed to cool to 70° F.—Urine, 45 ounces ; uric acid, ·700 grammes.
(3) Thermal water heated in closed vessel to 110° F.—Urine, 48 ounces ; uric acid, ·696 grammes.
(4) Ordinary drinking water at 110° F.—Urine, 47 ounces uric acid, ·696 grammes.

Bathing. (Over 100 cases.)

(1) At 82° F.—Urine, 55 ounces ; uric acid, ·939 grammes.
(2) At 90° F.—Urine, 50 ounces ; uric acid, ·874 grammes.
(3) At 96° F.—Urine, 47 ounces ; uric acid, ·777 grammes.
(4) At 100° F.—Urine 40 ounces ; uric acid, ·680 grammes.

(5) Bath in ordinary water at 82° F.—Urine 41 ounces; uric acid, ·675 grammes.

(6) Ordinary water at 100° F.—Urine 38 ounces; uric acid, ·669 grammes.

Drinking and Bathing at 82° *F.* (Over 100 cases.)

Average urine, 60 ounces; uric acid, 1·004 grammes.

While drinking the water, the quantity of uric acid excreted gradually reached its maximum in 14 to 20 days, kept about this figure for another seven or eight days, and then gradually declined to the normal amount.

This was also the rule when bathing in the water heated to 90° F. and over, but immersion in the water at its natural temperature (82° F.) was often followed by a considerable increase in the uric acid excretion even from the very first bath.

It will be noted that this bath contains the full amount of nitrogen gas undisturbed by heating.

The result of this investigation may be summed up as follows :—

(1) That the excretion of uric acid and the flow of urine are both largely increased by the drinking of, and by immersion in, the Buxton thermal water.

(2) That it is more efficacious when bathed in than when drunk as regards uric acid excretion.

(3) That the diuresis is somewhat greater when drunk than when used as a bath.

(4) That any deviation from the normal temperature either by heating or by cooling interferes with its efficacy.

(5) That while the alterations in temperature would not be likely to diminish the therapeutic properties of the salts they would on the other hand greatly interfere with the contained gas.

The late Dr. Robertson, for so long the leader of the medical profession in Buxton, and who did so much to press forward its claims as a gout spa, wrote :—

"It may be advanced that the modern researches of chemistry have caused more and more importance to be attached to the great elementary principle with which the Buxton tepid waters are surcharged. The very large proportion of nitrogen contained in the essential crystallisable principles of tea, coffee, and cocoa, in

taurine the great azotised constituent of the bile, in the various fibres and tissues, in the secretion and excretion of the body, the consumption of nitrogen shown to be involved in every movement and process of the animal systems, are so many indications of the inferences deducible from the great advance of modern chemistry, and so many reasons why this nitrogen in an available form for internal and external use may have a significance which may hardly admit of exaggeration. Argon being much more soluble than nitrogen, and therefore more rapidly absorbed through the skin and mucous membranes, appears to give an additional explanation of the influence upon the tissues of the body of these nitrogenous waters."

Dr. Thresh writes:—

"Doubtless much of the gas is liberated within the very pores of the skin itself during bathing, and acts in what may be considered its semi-nascent state, producing effects altogether unattainable by the use of the same agent in any other condition."

If it be true, as seems to have been proved beyond doubt, that the manifestations of gout are largely due to deficient excretion of uric acid, it is of importance that a natural water which so largely increases that elimination should have its therapeutic position placed upon a definite scientific basis.

Dr. Luff thought the paper was both instructive and suggestive. Mr. Armstrong's initial statement as to the diminished excretion of uric acid in the early stages of gout was very welcome to him personally. Although he was not by any means the first to point out that, contrary to the generally received opinion, there was a diminished excretion of uric acid before an attack of gout, yet he had laid special stress upon this fact in his Goulstonian Lectures two years ago. Formerly it was thought that the commencement of an attack of gout was associated with an excessive excretion of uric acid. The reason why this erroneous view had been so generally entertained was, no doubt, due to the fact that in the early stage of gout the urine was always laden with amorphous urates. The cause of that was to be found in the diminished excretion of urine in the early stages of gout which led to the urates being thrown down on the cooling of the urine. This, however, was no proof that the excretion of uric acid was increased; on the contrary, he had shown by numerous observations that the excretion of uric acid was invariably diminished. That was a point of great importance in view of an intelligent treatment of cases of gout. He commented on Mr. Armstrong's conclusion which showed that the results of his experiments with the free nitrogen, whether taken by ingestion or absorbed by the skin, apparently had a very remarkable

effect in increasing the excretion of fluid and of uric acid, and also to some extent of urea. He was quite prepared to learn that nitrogen, which had previously been regarded as an inert substance, might under certain circumstances exert a well marked effect, and that in its almost nascent state it might have a quite different effect to the nitrogen which was present in the atmosphere. His mind was the more open on that point seeing that a year since he had obtained some remarkable results in determining the solvent effects of vegetable ashes upon gouty deposits. When, however, he made an artificial ash based on the chemical analysis of the natural ash, which latter possessed such remarkable solvent properties, he had found that though the artificial ash was chemically identical with the other, yet it was quite unable to produce the same solvent effects. This made him very dubious as to the utility of deducing from the analysis of a natural mineral water what might be its therapeutical effects. We could imitate a mineral water and make it chemically indistinguishable from the natural one, but we could not necessarily imitate its therapeutic effects. From this he inferred that there were in these natural substances certain combinations which could not always be artificially reproduced, and such he imagined was the case with the waters to which Mr. Armstrong had drawn attention.

Mr. Armstrong, in reply, said he was glad to hear Dr. Luff's view because it held out hope of further progress in that direction. It was only since the work of Dr. Luff and the exact work of Gowland Hopkins that they had been enabled to determine the exact amount of uric acid excreted under such circumstances.

May 8th, 1899.

ENCYSTED VESICAL CALCULI, WITH AN ACCOUNT OF TWENTY-SEVEN CASES.

By W. Bruce Clarke, F.R.C.S.

In 1891* I published an account of six cases of encysted vesical calculi. Since that date I have had 13 other cases under my care, and, by the kindness of my colleagues at St. Bartholomew's, have been able to describe eight other cases, most of which I had the good fortune to see during their stay in hospital, making a total of 27 cases in all to form the materials for this paper.

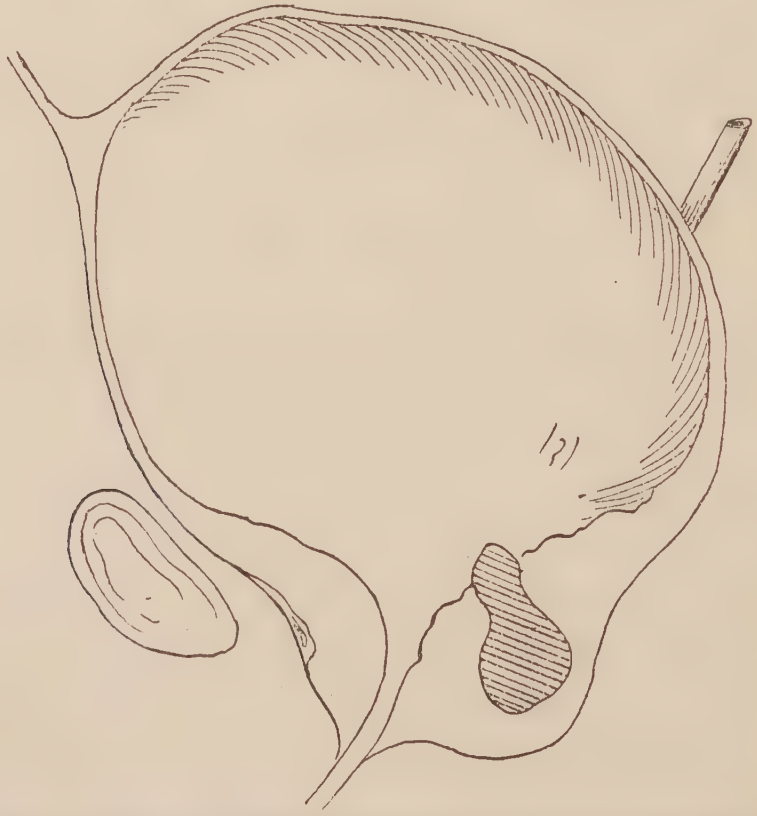
As I pointed out in my former paper, the ordinary symptoms that we are accustomed to associate with vesical calculus are frequently absent in such cases, and consequently the diseased

* "Latent Calculus in the Bladder," 'St. Bartholomew's Hospital Reports,' vol. xxvii, p. 117.

condition assumes oftentimes considerable proportions before it is detected. A stone which is fixed does not, as a rule, give rise to hæmaturia, and the acute pain, which is generally a consequence of the stone becoming grasped by the neck of the bladder, is usually remarkable for its absence. It may further be added that in some cases the stones are so deeply embedded in the bladder wall as to elude the beak of the sound, and only proclaim their existence when an attack of cystitis has supervened.

A perusal of the accompanying clinical histories cannot fail to convince anyone of the anomalous symptoms that are often present in such cases, and of the difficulties which must always beset their early recognition. It is at the same time a striking proof of the need of exploration in those cases of bladder disease which do not yield to the ordinary recognised modes of treatment.

Case 7.—J. J., aged 62, was sent to me in May, 1893, with history of four months' trouble with micturition. The prostate was slightly enlarged. A small amount of pus in the urine at times. May 15th: He



Case 7.—Shows the situation of stone (about one-fourth natural size).

was sounded, and a small stone detected on the left side of the base of the bladder. It was not easy to feel it, and it could not be seized with the lithotrite, or made to shift from its position. Suprapubic cystotomy

was accordingly performed. When the bladder was opened the small end of a calculus which was embedded at the prostate could be seen sticking up into it a little below the orifice of the left ureter, which appeared to open just at its margin. At first it seemed as if the stone could not be shifted at all, but by carefully moving it about with the finger it was eventually extracted, leaving behind a deep pit where it had lain concealed. The patient made an excellent recovery, leaving the hospital on June 19th. Two years later he came to see me, and on examining him with the cystoscope no trace of the sacculus could be detected. The stone in question is formed of uric acid. After descending from the kidney it had in all probability found its way into some depression on the floor of the bladder, and there gradually increased in size, and slowly enlarged the sacculus to suit the needs of its growth.

Case 8.—J. B., aged 67, first came under my care in February, 1893. He stated that seven years ago he had suffered from hæmaturia, and on one or two occasions since. For the last six months he had suffered a good deal of pain in the region of the bladder, and had frequent calls to micturition. His urine was foul, alkaline, specific gravity 1016, and contained a trace of albumen. His bladder was washed out frequently, but without affording him much alleviation. March 7th, 1893 : Admitted into hospital ; sounded with a negative result. As he did not improve, on March 23rd suprapubic cystotomy was performed. Deep down behind the prostate, and apparently almost outside the bladder, was found a stone of peculiar character. Its inner part was composed of oxalates, and it lay in a shell of phosphates, which had begun to redissolve, so the original oxalate nucleus was surrounded by a loosely-fitting crystalline phosphatic shell. In the process of extraction the shell was somewhat damaged. He recovered from the effects of the operation, and was able for awhile to get up and get about the ward ; but the water remained foul and alkaline in spite of careful washing out of the bladder. Early in May of the same year he took to his bed again, and, getting gradually worse, sank from severe hæmorrhage on May 11th. The *post-mortem* examination demonstrated what was suspected at the time of operation, namely, that the pouch had practically perforated the bladder. The vesiculæ seminales contained pus, and some pus was also found in the cellular tissue round the base of the bladder. The kidneys were not diseased.

Case 9.—J. P., aged 66, came under my care with all the ordinary signs of enlarged prostate, complicated by cystitis. He said he had been quite well up to two months previously. He was admitted to St. Bartholomew's Hospital on July 10th, 1893. His bladder was regularly washed out, but he did not make much progress. August 5th : He was sounded and examined with the cystoscope. The bladder would contain only 4 ounces of water. No satisfactory view could be obtained, and no evidence of stone was discovered with the sound. August 7th : Suprapubic cystotomy was performed, and two stones were found in a cyst behind the prostate. There was a well-marked prostatic enlargement at the entrance of the bladder, which was partly cut off and partly burnt away with a cautery. His recovery was prolonged but uneventful. The suprapubic wound took a long time to heal, and he did not leave the hospital until November 24th. At this time most of the urine had to be passed by catheter.

Subsequent History.—He has been seen on several occasions since ; the last time rather less than a year ago. He has regained almost complete power of natural micturition, but usually passes a catheter before going to bed, and occasionally washes out his bladder. He states that there is often no residual urine.

Case 10.—J. P., aged 68. (Sent to me by Dr. Lancelot Rolleston.) November 7th, 1893. He stated that some four or five years ago he had some irritability of his bladder, and applied to a surgeon in New Zealand, where he was living at the time. He was sounded, but no stone was found. His symptoms disappeared almost entirely for several years. In 1889 he was again sounded by the same surgeon and a stone discovered. He came to England and placed himself under the care of a surgeon who had had a very large experience of such cases. His stone was crushed, but though he remained some months longer in England the symptoms never quite disappeared. He was assured by the surgeon who had operated on him that there was nothing more in his bladder, and that by the time he reached New Zealand he would be quite well

Case 10.—Two facetted stones removed from pouch behind prostate (slightly less than natural size).

This did not prove to be the case, and in 1893 he placed himself under my care. I sounded him on two occasions, and on the second the sound seemed to touch a rough surface on the bladder wall. I advised suprapubic cystotomy, and on November 26th, Dr. L. Rolleston being present, the operation was performed, and two facetted stones were removed from a pouch behind the prostate. Their upper surface was smoothed, presumably by the stone which had been removed by lithotrity two years earlier. On January 9th, 1894, he sailed for New Zealand, where he has enjoyed excellent health ever since. He was last heard of in November, 1898.

Case 11.—A. C., aged 64, was sent to me by the late Mr. Morrant Baker, under whose care he had been on several occasions on account of some prostatic trouble of several years' duration. In March, 1893, when I first saw him, he was complaining of an aggravation of his symptoms, and I sounded him and touched a calculus on the back wall of his bladder. An operation was advised, and on March 18th suprapubic cystotomy was performed, Mr. E. Willett giving the anæsthetic. He was sounded again at the time of operation, but no stone could be felt. His condition, however, seemed to warrant an operation. When the bladder was opened a stone was, after some little difficulty, found imbedded in the back wall of the bladder. Its free surface was so covered with inspissated mucus, the result of a recent attack of cystitis, that it was quite easy to under-

stand how it was that the sound had failed to detect its presence a few minutes previously. It was eventually removed from the sac in which it was embedded, but I must confess with no small misgivings on my part as to the future of the case, as I feared lest an attack of peritonitis should supervene. The sac wall was quite loose, and when the stone was extracted it easily turned inside out and bulged into the interior of the bladder. Under these circumstances I seized it with a pair of pressure forceps, which were left on for 48 hours and then removed. Barring some trouble that was experienced with his bowels, which may have been due to a slight attack of peritonitis, he made an excellent recovery, and left my hands about five weeks later. I saw him a year afterwards in excellent health. The stone was mainly composed of uric acid, but the surface which lay exposed in the interior of the bladder is coated with a layer of phosphates.

Case 12.—J. G., aged 71, came under my care in December, 1894. He was admitted to the hospital on December 7th. He looked very ill and emaciated.

History.—He had had urinary trouble off and on for 14 years. Six years before he underwent lithotrity at St. Peter's Hospital. Four years before he underwent a second operation at St. Peter's. Two years before he had a third operation at St. Peter's, and was informed that two stones had been crushed. On each occasion he was considerably benefited, but since the last operation had been obliged to pass most of his water by catheter. On December 9th he was sounded by me, and a stone was felt in the prostatic urethra. A lithotrite was introduced and a small stone was crushed and washed out, but the remainder could not be seized, and appeared to be fixed to the bladder wall. Under these circumstances I decided to perform suprapubic cystotomy, and opened the bladder without any difficulty. Three stones were found firmly encysted in the bladder wall some little distance behind the prostate, and two small ones were lying loose on the top of the others. One of them is smooth and not coated by rough phosphatic material like the others. In all probability it had only recently come down from the kidney. The other four were thickly coated with mucus when they were first extracted. The patient grew gradually weaker, and died on December 11th.

Post-mortem Examination.—Both kidneys were in a condition of pyonephrosis. There was no peritonitis or sign of injury to the bladder wall.

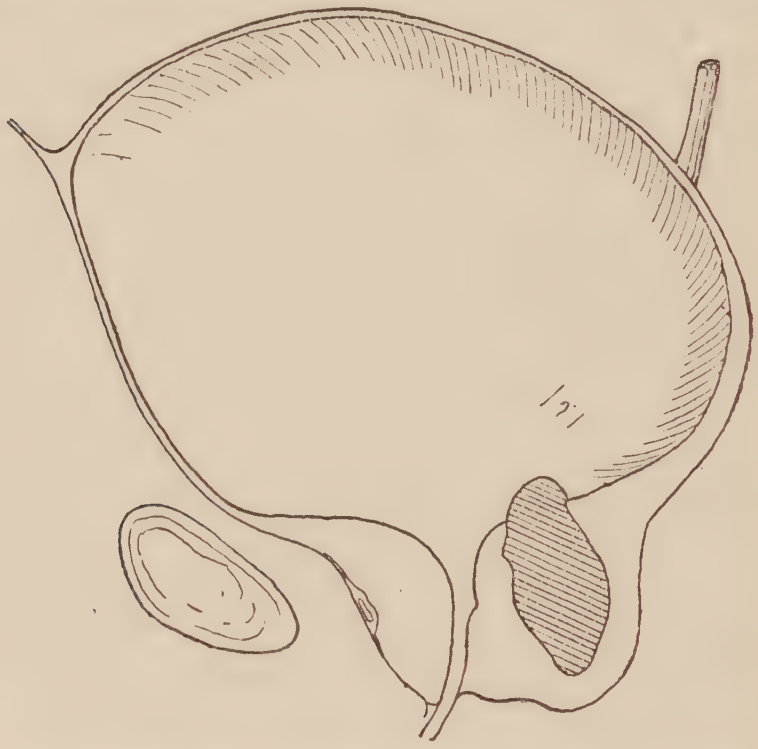
Case 13.—General C., aged 60, first consulted me in 1890 on account of some bladder trouble. He had had a stricture, and had been told his prostate was enlarged ; but as he was able to empty his bladder as a rule he was loth to make use of a catheter ; and though he had had repeated slight attacks of cystitis he declined to submit to any treatment. I only saw him on two occasions, and then lost sight of him until I heard of his death in 1894 under the care of Dr. Jowers, of Brighton, from peritonitis, which had been set up by the ulceration of a stone from a pouch on the back wall of his bladder into his peritoneum.

Case 14.—June 20th, 1894. Colonel C., aged 80 (sent me by Dr. G. Coates, of Kensington), was suffering from an enlarged prostate, and had used a catheter at times for seven years. He had lately had to use it a good deal more often, and to get up frequently at night. His bladder

held about 4 ounces of acid urine, 1021 ; a trace of albumen. As he declined at first to undergo a complete examination, his bladder was washed out daily and he was treated with alkalies and diluents, but he did not improve. July 2nd : He was sounded under an anæsthetic, and a stone just touched behind the prostate. An unsuccessful attempt was made to seize it with a lithotrite, and suprapubic cystotomy was performed. A stone was found tightly fixed to the wall of a pouch behind the prostate and removed. His recovery was a prolonged one, but he left London on September 2nd, and a month later was able to go out shooting. He remained perfectly well for two years when an attack of cystitis supervened, and he died at San Remo.

Case 15.—J. S., aged 69.

History.—He first attended in my out-patient room in 1894. He stated that for three years past he had had trouble with his water. He had



Case 15.—Shows stone embedded in prostate (size about one-fourth).

been obliged to get up at night several times to pass water, and had frequent calls to micturate during the daytime. A catheter was passed, and he was found to have 6 ounces of foul residual urine. The prostate was considerably enlarged *per rectum.* He was instructed to wash out his bladder regularly, and to draw off his residual urine before going to bed ; and under this line of treatment he rapidly improved, and his urine became quite natural. About a year later (1895) his symptoms returned in an aggravated condition, and he was admitted into the hospital. Considerable difficulty was experienced in introducing a catheter, and no amount of washing out could make his urine healthy. His bladder was carefully examined on more than one occasion for stone, but none could be discovered. The cystoscope was also introduced, but no clear

view could be obtained even after frequent washings out. The enlargement of the prostate rendered the passage of an instrument an exceedingly difficult procedure, and he was quite unable to pass any instrument for himself. Under these circumstances castration was advised, and he consented to the operation. It was performed on June 29th and he sank gradually three days later.

A *post-mortem* examination was made on the following day. The kidneys displayed well-marked interstitial nephritis. Acute cystitis was present, and embedded behind the prostate was found an encysted calculus. It was contained in a pouch which only communicated with the bladder by a very small aperture. The tip of the stone protruded into the bladder, and was thickly coated with mucus. It is doubtful whether even this slight protrusion was not due to the shrinkage which had already begun to take place in the prostate.

Case 16.—J. T., aged 57.

History.—He stated that he had had a stricture for 20 years, and had had to have his water drawn off from time to time. Six years ago he was thrown out of a cart, and injured his perineum, since which time his urine had had to be drawn off by catheter.

Present Condition (April 10th, 1895).—No. 6 passes to bladder. Two strictures, one $2\frac{1}{2}$ inches from meatus and one in the deep urethra; urine 1010, alkaline, offensive; contains pus. His bladder was washed out daily, but he died 10 days later. He was at first under Mr. Langton's care, but I saw him, in Mr. Langton's absence, on two occasions before he died.

Post-mortem Examination.—Signs of peritonitis, and some recent subacute peritonitis as well. The adjacent viscera were firmly adherent to the bladder. The kidneys showed signs of interstitial nephritis; the ureters were not dilated. Bladder hypertrophied and dilated, and contained nearly two pints of fluid. At the upper left side there was a considerable pouch, from which the peritonitis had started. Its interior was very ragged from ulceration and sloughing of the mucous membrane. There were two calculi in the pouch. There was no direct opening into the peritoneum.

Case 17.—R. W., aged 64, was admitted to St. Bartholomew's Hospital, under Mr. Butlin's care, suffering from hæmaturia and frequent micturition. August 2nd, 1896: A severe attack of hæmaturia supervened, and, being on duty for Mr. Butlin, I was summoned to see the case. His bladder was almost full of clots, which could not satisfactorily be washed out with an aspirator, and he was obviously suffering from cystitis. Under these circumstances I decided to perform suprapubic cystotomy without delay. The bladder was opened, and found to be filled with clotted blood and highly-offensive urine. When this had all been cleared away two stones were found lying in a pouch behind the prostate, from which they were only extracted with difficulty. One was about the size of a filbert, and the other somewhat smaller. A pedunculated middle lobe of the prostate was found and removed. The cause of the bleeding was not clearly determined. It was probably due to the congested piece of prostate, which was removed, and which appeared to have had its pedicle partially twisted. He was discharged about six weeks later, able to micturate usually without the aid of the catheter. He was in the habit of washing out his bladder daily. Three months later I saw him

again, and he told me he felt perfectly well, and only used the catheter once a week as a matter of precaution.

Case 18.—C. S., aged 64. October 30th, 1897.

History.—Two years ago he began to have pain and difficulty in micturition, which soon became very bad at night. In John Ward for cystitis. Bladder examined with cystoscope with negative result. Bladder washed out regularly, and some improvement occurred.

Present Condition.—Emaciated. Complains of great deal of pain over region of bladder. Urine 1014, alk. albumen, pus, triple phosphates, ammoniacal, and fœtid. Residual urine ʒviij. Bladder washed out without much result. Cystoscopic examination negative. The urine was so thick that no satisfactory examination could be obtained.

November 22nd: Patient placed in Trendelenburg's position, and perineal cystotomy performed. A vaginal speculum was passed in through opening, but no good view could be obtained of interior of bladder. February 9th: A second examination made of the interior of bladder, as the patient's condition had scarcely improved at all. Patient gradually sank and died seven days later.

Post-mortem Examination.—A large sacculus was found in connection with the bladder on its back wall. The sacculus held, when distended, nearly half a pint of fluid. The opening by which it communicated with the interior of the bladder was circular and about $\frac{2}{3}$-inch in diameter, and closely adjacent to the orifice of the left ureter. The sacculus contained two stones coated with phosphates, one about the size of a walnut, and the other quite a small one. There was suppurative nephritis of both kidneys.

Case 19.—J. P., aged 67. August 3rd, 1898.

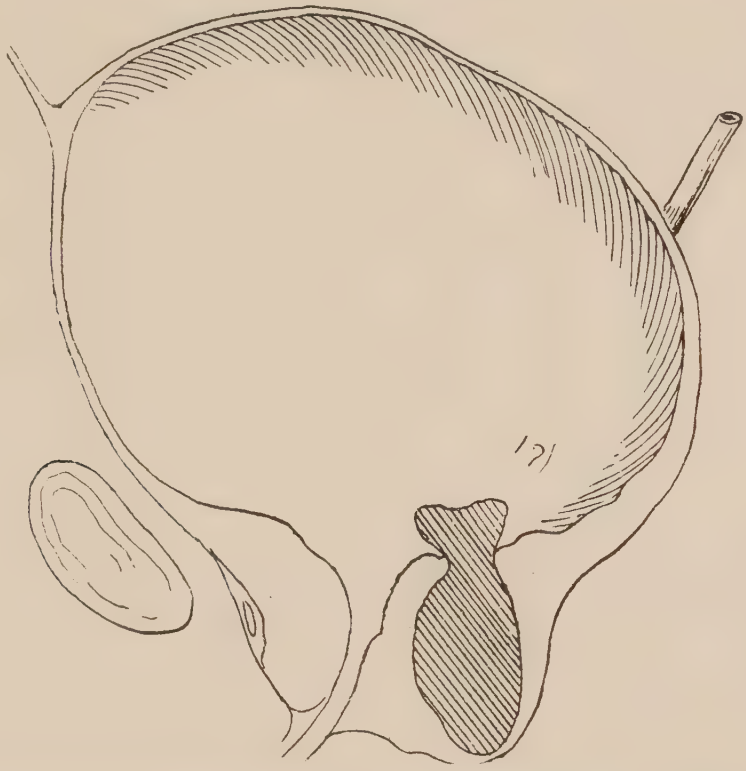
History.—Three years he had had trouble with micturition. He had been getting steadily worse lately. He had lately been unable to pass his water without catheter. Urine foul, ammoniacal, specific gravity 1015, contains pus and albumen. Prostate enlarged; has his water drawn off twice daily; amount varied very considerably. August 9th: Cystoscopic examination revealed general cystitis. Drainage advised; patient's friends would not consent till he was too bad to undergo operation. August 13th: Died.

Necropsy.—Bladder small and contracted. Large saccule containing upwards of 2 pints of fœtid urine. It communicated with the general cavity of the bladder by a small hole just above the trigone, which would just admit the index finger. It contained five small phosphatic calculi. Ureters and kidneys full of pus.

Case 20.—J. E., aged 71.

History.—He had a stone crushed at the Middlesex Hospital in 1874. Remained free from all trouble until early in 1897. In May, 1897, he was admitted, under the care of Sir T. Smith, into St. Bartholomew's Hospital, with cystitis, and was twice sounded and examined with the cystoscope, but without anything being discovered to account for his cystitis, with the exception of some enlargement of the prostate. He was accordingly instructed to wash out the bladder. The following year, October, 1898, he came to my out-patient room with his urine in a very foul condition. His prostate was very hard and tender. Before arrangements could be made for his admission to the hospital, he took to his bed

and died a few days later. I obtained permission to examine his bladder, and found a large stone almost buried in a retroprostatic pouch. The lower part, which was embedded in the prostate, consisted of uric acid, whilst the upper part, which was contained in the bladder, was shaped like the calyx of a flower, and was composed partly of uric acid and partly of friable phosphates. Extreme prostatic tenderness was obviously to be accounted for by the position of the stone.



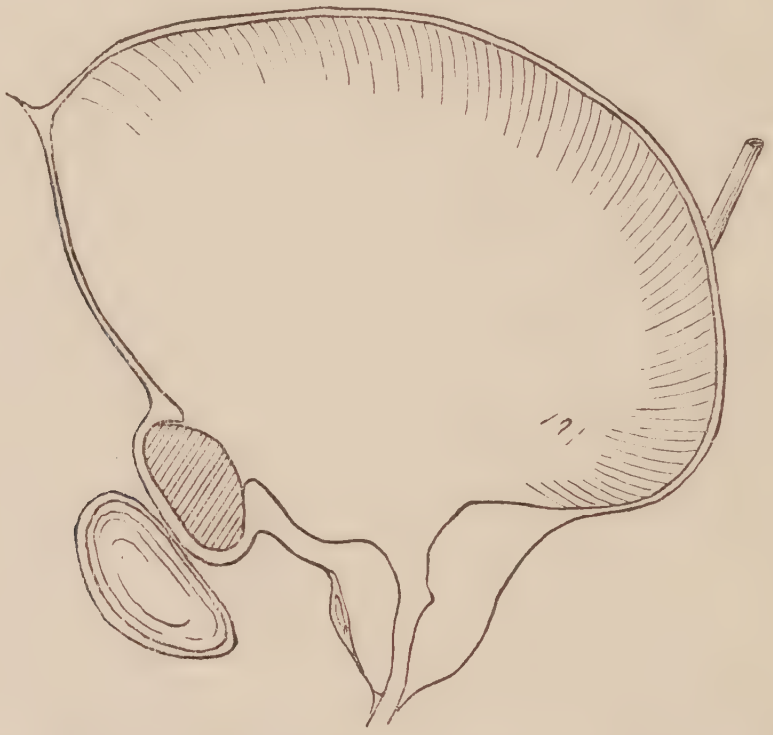
CASE 20.—Shows large stone, which had almost perforated the prostate. The stone is drawn a little smaller than it should have been. It was nearly 2½ inches in its longest diameter.

CASE 21.—F. P., aged 49 (under Mr. Langton and Mr. Cripps).

History.—He had had attacks of irritability of the bladder for many years. These attacks were at first attributed to a stricture. In 1887 he passed a small calculus, and a few months later he was admitted under Mr. Langton's care. Mr. Langton removed a stone by lithotrity, but the symptoms referable to his bladder still persisted. In Mr. Langton's absence, Mr. Harrison Cripps performed suprapubic lithotomy, and removed a stone from a sacculus which lay above the urethral opening behind the pubes. In September, 1892, some more fragments were removed by lithotrity, amounting to about 1¼ ounces. In November, 1892, suprapubic cystotomy was again performed by Mr. Harrison Cripps, and another stone removed from the sacculus above referred to. I was present on this occasion, and was able to look into the sacculus in question. The patient made an excellent recovery.

CASE 22.—W. W., aged 60 (under the care of Mr. Butlin). He was admitted into St. Bartholomew's Hospital on November 5th, 1897, with symptoms of great irritability of the bladder; was unable to hold his water more than a few minutes. His prostate was somewhat enlarged.

He stated that his bladder had been very irritable for several years past, water often thick, urine alkaline, containing pus. He was sounded on the following day, and a stone (apparently of large size) was detected lying behind the prostate, but it could not be grasped by the lithotrite or moved from where it was lying. Under these circumstances suprapubic lithotomy was performed, and a stone was found lying in a sacculus and tightly gripped by its walls, so that it was only after some difficulty that it was dislodged by the aid of a scoop. Convalescence uneventful. Patient left the hospital on December 16th, passing all his water *per urethram.*



Case 21.—Shows a sacculus in an unusual situation—semi-diagrammatic (size about one-fourth).

Case 23.—W. B., aged 57 (under Mr. Butlin's care). He came under Mr. Butlin's care in November, 1893. His bladder was examined by means of a cystoscope and with a sound. An epithelioma occupied its base, and an encysted stone occupied the left side of the base in close proximity to the orifice of the left ureter. The patient was very ill, and refused operation. He died a few days later, and the condition was verified at the *post-mortem.*

Case 24.—G. E. (under Mr. Butlin's care). He was admitted into St. Bartholomew's Hospital on April 6th, 1894. Urine foul, alkaline, contained a trace of albumen and some pus. He had been sounded before admission, and a stone detected. An attempt was made to perform lithotrity, but unsuccessfully, as the stone was encysted. The bladder was inspected with the cystoscope, and the stone clearly seen embedded in the back wall of the bladder. I was present at the examination and saw the stone. It was decided to perform suprapubic lithotomy a day or two later. In the meanwhile, the man discharged himself from the hospital.

Case 25.—J. B., aged 40. (Under Mr. Walsham.)

History, 1893.—Three years ago he had a stone crushed at St. Bartholomew's Hospital. February, 1896: For two years after last operation was quite free from all vesical symptoms, but has had some irritability of the bladder more recently. June 17th, 1896: The patient was sounded, but no calculus could be detected. A rectal examination revealed some considerable tenderness about the prostate. As this condition did not disappear by rest in bed, &c., Mr. Walsham performed median lithotomy five days later, and removed 10 stones, which were tightly wedged together in a pouch behind the prostate. A month later the patient left the hospital perfectly well, and there is a note in Mr. Walsham's own handwriting:—"The rectum is natural; the prostate feels small and harder than normal."

Case 26.—R. W., aged 38 (under Mr. Walsham's care). September 10th, 1894. He was admitted into St. Bartholomew's Hospital suffering from occasional hæmaturia and frequency of micturition; was sounded and a calculus detected. September 12th: Lithotrity. After a considerable amount of *débris* had been crushed and removed, some portions of stone remained behind which appeared to be firmly fixed to the bladder wall, and could not be readily seized with the lithotrite. Suprapubic lithotomy was performed, and on the left side of the base of the bladder a pouch was discovered. A stone was sticking out of it, the top of which had been removed by the lithotrite. It was extracted after some difficuly with lithotomy forceps, and a second stone was found lying in the same pouch and was removed. The patient made a good recovery, and left the hospital well on October 25th, 1894.

Case 27.—May 10th, 1893. H. B., aged 14.

History.—Six years ago lithotrity was performed by Mr. Willett. Four years later the operation was repeated by Mr. Willett. One year ago Mr. Marsh removed a stone from a distinct pouch behind the prostate after suprapubic cystotomy. May 12th: Mr. Bowlby sounded the patient, and found a stone firmly wedged at the neck of the bladder, and could not seize it with a lithotrite. He accordingly performed the suprapubic operation, and removed an oxalate stone partially coated with phosphates, which was embedded in the same pouch from which Mr. Marsh had removed the previous stone.

Remarks.

Of the six cases that were recorded in my first paper, no fewer than three were due to pieces of bone that had found their way into the interior of the bladder from the surrounding structures. The other cases were instances of true encysted calculi, which had been formed presumably in the kidney, and then, finding their way into the bladder, had either dropped into a sacculus which was ready for their reception or had become fixed in some depression of the bladder wall, and had by their gradual growth formed a sacculus for themselves. It is probable that in at least one of the cases referred to in this paper—namely, No. 7—

this was the way in which the calculus was formed; for when this bladder was inspected with a cystoscope two years after the removal of the stone no trace of a sacculus was to be seen. In Case No. 3,* on the other hand, the sacculus was most likely quite independent of the stone, for stones were found on a second occasion in another part of the bladder, and the sacculus originally occupied was empty.

It has often been assumed that these sacculi are all due to what are called tunicary herniæ, and depend on the pouching of the mucous and submucous coats between the bands of muscular fibres. I am, however, convinced, after an examination of numerous specimens both before and after death, that the muscular walls very often form a constituent part of these sacculi. It is, moreover, often very difficult to determine with accuracy in all cases the nature of the sac walls owing to the inflammation which has been set up by the stone which has long lain there.

When operating on these cases there is one point which must always be considered, namely, what is to be done with the empty sac from which a stone has been removed. There is no doubt that many of these sacs, especially when prolonged attacks of inflammation have destroyed their epithelial lining, tend to contract and cicatrise of their own accord if their orifice is of sufficient size to preclude the possibility of pus stagnating in their interior. If, on the other hand, the orifice of the sac is small and contracted and the sac itself of large size, some means must be adopted for its independent drainage, and the route which is selected must depend on the condition of the patient and the position of the sacculi. As most of these saccules are situated near the base of the bladder, it will probably be advisable if they demand drainage to drain them through the perineum, and to make a temporary channel through the perineum in front of the rectum if this is possible.

On looking over the symptoms which these cases presented it is difficult to single out any one sign which might be held to be of considerable diagnostic value. Cystitis of longer or shorter duration appears to have been present in every one of the cases, but as the great majority of them—namely, 16 out of 21—were above 60 years of age, and had in addition some evidence of

* *See* previous paper, *op. cit.*

enlargement of the prostate gland, the cystitis was naturally in the first instance ascribed to that condition, and not to the presence of a calculus. In several undue tenderness of the prostate gland when examined *per rectum* and rectal tenesmus were marked symptoms. Only three of the cases were under 40 years of age, one of them being but 14. The only two cases in the list between the ages of 50 and 60 had suffered from stricture; and it is noteworthy that two of my first six cases also had suffered from stricture, and were likewise between 50 and 60 years of age.

The difficulties of diagnosis were also enhanced by the fact that in several instances washing out the bladder was quite sufficient temporarily to restore the urine to a healthy condition. It is, perhaps, also worthy of note that in several instances the stones when extracted proved to be so thickly coated with mucus, that even when they lay upon the table no audible note could be obtained when they were struck with the beak of the sound. This affords a ready explanation of the difficulty which may be experienced in detecting a stone in the bladder under certain conditions.

In six of the 21 cases no operation was performed in most instances, because the patient's condition was too bad to admit of any chance of alleviating his symptoms. Of the remaining 15 that were submitted to operation four died and 11 recovered.

The fact that in less than half the cases was the existence of the stone known before the operation was undertaken, certainly affords an additional argument for exploration of the bladder in those cases of cystitis which do not yield to ordinary treatment.

In one instance (namely, Case 15) the patient had been castrated to relieve his prostatic symptoms, and the stone was only detected after death. It is quite possible that if his bladder had been explored at an earlier period he might have retained his testes and had his life prolonged after removal of the stone.

I have since that time made it a rule to explore the bladder in obscure cases of enlarged prostate which remain irritable after repeated washing, and have never had occasion to regret this plan of treatment, which I have no doubt largely accounts for this series of encysted calculi. Even if no stone is detected the patient usually derives great benefit from the operation, owing to the rest which is afforded to the urethra.

Table of Cases.

Number.	Patient.	Surgeon.	Prominent Symptoms.	Result.
1	W. M. (55)	Bruce Clarke	Stones removed on two occasions successfully. Died 3 years later.	R.
2	E. H. (67)	,,	Stricture. Encysted stone found at *post-mortem* examination.	D.
3	H. H. (57)	,,	Stricture; stones removed from cysts on several occasions.	R.
4	—	,,	All dependent on pieces of bone that had protruded into bladder and become nuclei for stones. One died.	D.
5	—	,,		R.
6	—	,,		R.
7	J. J. (62)	Bruce Clarke	Frequent micturition: some pyuria ...	R.
8	J. B. (67)	,,	Pain; hæmaturia; foul urine	D.
9	J. P. (66)	,,	Cystitis; enlarged prostate...	R.
10	J. P. (68)	,,	Pain and cystitis two years	R.
11	A. C. (64)	,,	Prostatic trouble several years	R.
12	J. G. (71)	,,	Symptoms 14 years; lithotrity on two previous occasions.	D.
13	Gen. C. (60)	,,	Repeated slight attacks of cystitis. No operation.	D.
14	Col. C. (80)	,,	Enlarged prostate 7 years; catheter ...	R.
15	J. S. (69)	,,	Enlarged prostate 4 years; castration ...	D.
16	W. J. T. (57)	Self and Mr. Langton	Stricture 6 years. No operation	D.
17	R. W. (64)	Self and Mr. Butlin	Severe hæmaturia	R.
18	C. S. (64)	Bruce Clarke	2 years cystitis off and on	D.
19	J. P. (67)	,,	3 years cystitis. No operation	D.
20	J. E. (71)	,,	Stone crushed 22 years before death; cystitis 1 year. No operation.	D.
21	F. P. (49)	Mr. Langton and Mr. Cripps	Stricture; recurrent attacks of cystitis ...	R.
22	W. W. (60)	Mr. Butlin	Recurrent attacks of cystitis	R.
23	W. B. (57)	,,	Stricture; cystitis some months and epithelioma of bladder. No operation.	D.
24	G. E. (61)	,,	Cystitis. Left hospital	*
25	J. B. (40)	Mr. Walsham	Lithotrity 3 years previously; cystitis 3 months.	R.
26	R. W. (38)	,,	Hæmaturia and cystitis for several months.	R.
27	H. B. (14)	Mr. Willett and Mr. Bowlby	Lithotrity twice; lithotomy twice within 18 months. Stone removed from retro-prostatic pouch.	R.

* The result of Case No. 24 is unknown.
The Cases Nos. 7 to 27 are referred to in the present paper.

The PRESIDENT expressed surprise at the frequency with which in the cases that had been narrated the use of the endoscope had given a negative result. He urged that in obscure cases of cystitis which did not improve on washing out, suprapubic exploration was of the greatest possible value, and that the use of a vaginal speculum through the suprapubic opening greatly facilitated the vesical examination.

Mr. SWINFORD EDWARDS remarked that the author seemed to have had an unique experience of encysted calculi. They probably did not have more than 10 cases of stone a year at St. Bartholomew's Hospital, yet the author had been able to bring together 25 cases of encysted stone within the last few years. He said that he himself had a personal experience of some 800 cases of vesical calculus, either in his own practice or in that of his colleagues, and among these there were very few cases of encysted

calculi. Personally he had only had two such. It would appear consequently that at St. Bartholomew's they must have had an undue proportion of these cases.

The PRESIDENT pointed out that some of the author's cases were met with in private practice.

Mr. EDWARDS, continuing, said that he had been surprised that better results had not been obtained with the cystoscope in the cases in which it had been possible to introduce the instrument. With an enlarged prostate its introduction was not always possible ; and, even if this were accomplished, there was apt to be much bleeding, which obscured the field of vision. He himself had not experienced so much difficulty with the cystoscope, which was indeed a very valuable aid to diagnosis. He was glad to hear the author advocate suprapubic cystotomy in cases where encysted calculus was diagnosed. Some surgeons attempted to crush the stone *in situ*, or at any rate to seize it and drag it into the bladder. Such a procedure he thought was fraught with much risk. He had done it once himself in a female who died of septic peritonitis, and this experience had determined him never again to attempt to crush a calculus in a cyst. On another occasion he had had to deal with a large calculus weighing nearly 3 ounces, lying in a pouch above the pubes. With the lithotrite he was unable to dislodge it or to succeed in crushing it *in situ*, so he had to do a median perineal cystotomy. He was then able to dislodge the stone from the suprapubic region, and to bring it into the bladder where he crushed it by means of Bigelow's largest lithotrite. He urged the importance in these cases of not persisting in the attempt to crush, but at once to do suprapubic cystotomy. The cyst, if necessary, might be drained through the perineum.

Mr. BUCKSTON BROWNE observed that the paper was one of the most interesting and important they had listened to for some time. He agreed that the number of these cases which the author had been enabled to bring forward was remarkable, but he thought this admitted of explanation. He asked the author to explain the title of his paper and to define the meaning of sacculated calculus. His own definition would be "a calculus embedded in the walls of the bladder," but there were at least five of the author's cases which would not answer to this description, the calculi being in a post prostatic pouch. Encysted calculi might be divided into sacculated and post prostatic, and it was very important to distinguish these two varieties because the surgery and the results of the two were very different. In the author's cases they would find that the fatalities occurred among the true sacculated calculi. A stone in a post prostatic pouch could be removed by suprapubic operation without any laceration of the bladder wall, but it was very rare for one to be able to get away a true sacculated stone without injury to the bladder, and consequent risk of complications. He raised the question as to what ought to be done to these sacculi after removal of the stone, and he referred to a remarkable case in his own series in which he had had three times to perform suprapubic cystotomy in order to move a calculus from a sacculus, which could only be done by this means. He concurred in what had been said with regard to suprapubic cystotomy ; in fact, all the knowledge they possessed of the subject was derived entirely from and through the suprapubic incision, such cases being previously overlooked. The surgery of encysted calculi was entirely modern, and in many cases immense good could be done by performing the suprapubic operation.

Mr. P. J. FREYER said the best proof he could give the author of his

appreciation of the subject was that he had contemplated writing a paper thereon himself. He said he had now operated upon nearly a thousand cases of stone in the bladder, and, although he had searched for encysted stone very carefully, he had only come across 20 or 25 cases in the whole number. He agreed with the previous speaker that a stone lying in a post prostatic pouch should not be regarded as an encysted calculus. Wherever there was an enlarged prostate, the bladder cavity became divided into two, an anterior and a posterior, and a stone would of course lie most frequently in the posterior cavity. It would not, however, necessarily be encysted, though in some of the author's cases the stone appeared to have been encysted in that situation. The pouches might be divided into two kinds according as the opening by which they communicated with the general bladder cavity was small or large. When the stone was encysted in a pouch with a narrow opening, a cutting operation of some kind became indispensable preferably by the suprapubic operation ; but when the opening was a wide one his own practice was to crush the stone. With regard to the proportion of cutting operations to the total, he mentioned that of the last 400 operations performed on patients whose ages varied from 18 months to 90 years with calculi varying in weight from a few grains to $6\frac{1}{4}$ ounces, only eight had necessitated a cutting operation. If a stone were encysted on the posterior wall of the bladder there might be no hæmorrhage, but the most severe case of hæmaturia which he could remember was in a patient with a stone in this situation who, when he went to stool, experienced violent pain due to pressure on the stone.

Mr. Bruce Clarke, in reply, said that when he was connected with St. Peter's Hospital he believed he did not once see an instance of encysted calculus, but it often happened at special hospitals that the patients diagnosed their own complaints, whereas those who went to the general hospitals had theirs diagnosed for them. It followed that many obscure cases found their way to the general hospitals. He had noticed that in respect of many other complaints, ovarian disease for example. When he compared the cases seen at the Samaritan Hospital with those at St. Bartholomew's Hospital, the latter were much more complicated. The same held good in respect of eye cases, and they would see a greater variety of obscure cases of eye disease in a general hospital than at Moorfields. As to the cystoscope, he pointed out that in many instances it was not of much use, because of the bleeding ; again, many of the stones were not visible even when they had opened the bladder, they could not even be felt. Then, too, when the bladder was so contracted as not to hold enough urine a good view was impossible. He agreed with Mr. Freyer in respect of crushing stones in sacculi, that there were circumstances and circumstances, sacculi and sacculi. There were some in which the lithotrite could be introduced and the stone crushed and washed out, but no one would suggest that this procedure should be resorted to indiscriminately. Mr. Buckston Browne had alluded to sacculated calculi, by which he presumed was meant sacculated bladder. His own definition of an encysted calculus was one which was contained in a definite cavity of adventitious origin ; in other words, they must distinguish between a pouch and a sacculus. He believed that all the stones he had shown that evening were actually encysted either in the substance of the prostate itself or in a definite diverticulum of the bladder, and were not contained in the post prostatic pouch. His own impression was that some of these stones were formed round nuclei which were met with in the prostate

gland, and he referred to a case which he had shown some years ago which supported that view. His object in bringing forward the paper was that they might realise that there were a great many cases of stone which it was almost impossible to diagnose without opening the bladder, their knowledge whereof had entirely arisen since the reintroduction of the suprapubic operation. He thought that, in cases of doubt, at an early stage they should advise an exploratory operation.

TWO CASES OF RESECTION OF THE LARGE INTESTINE, WITH RECOVERY.

By Frederic Eve, F.R.C.S.

These two cases are examples of the commonest causes for which the large intestine may require resection—namely, for strangulation and gangrene by hernia, and for carcinoma. They both illustrate the wisdom of postponing resection when obstruction exists until this has been relieved. Case 2 is published especially to draw attention to the merits of extra-peritoneal resection and suture for artificial anus.

Case 1. *Strangulated Left Inguinal Hernia; Herniotomy; Perforation of Transverse Colon; Resection of Bowel Five Weeks Later; Recovery.*—T. T., aged 46, was admitted to the London Hospital on November 23rd, 1896, with a large, strangulated, left scrotal hernia. The hernia had existed for 20 years, and he had worn a truss. On November 20th the rupture came down, and he was unable to reduce it. Vomiting commenced on the 22nd inst. The bowels had not acted since the 19th. On November 23rd herniotomy was performed. The sac contained omentum and a portion of the transverse colon. This was deeply congested and perforated; the opening was too large, and the condition of the bowel too unsatisfactory to admit of closing the opening by sutures. A glass tube (Paul's) was tied in the large intestine, and this was fixed by one or two sutures to the skin in the inguinal region. The patient recovered from this operation without any troublesome complications. A portion of bowel covered with granulations protruded from an opening in the abdominal wall, and the whole of the fæces were discharged through it. After allowing an interval of five weeks to elapse in order that the parts might consolidate and the inflammatory thickening disappear, the operation of resection was performed on January 2nd, 1897. Anæsthetic, ether, and A.C.E. mixture. An incision was made vertically upwards in the semilunar line from the small protrusion of the bowel. This divided all the structures down to the peritoneum. It was my intention to perform the resection entirely extraperitoneally, after the manner suggested by the late Mr. Greig Smith.* But the peritoneum was found so thin and delicate that it would not permit of the necessary manipulation. This was doubtless due to stretching of the membrane

* 'Abdominal Surgery,' vol. ii, p. 634.

from the proximity of the hernia. The peritoneal cavity was therefore opened, the proximal end of the bowel secured, and the protruded portion then separated by the division of the abdominal parietes around it. The knuckle of intestine to be excised was brought quite out of the abdominal wound, and the peritoneal cavity protected with sponges. The gut was resected, and the divided mesentery brought together with a running suture. The mucous membrane of the free ends of the intestine was first united by a continuous suture of fine silk. Next the peritoneal and muscular coats were united by about 50 Lembert sutures. The running suture through the mesentery was tied last. Finally, a graft of omentum was fixed around the line of sutures, especially near the mesenteric attachment. The abdominal wound was closed, and a small drain was left in it. This was removed in 24 hours. The operation lasted about two hours. The patient suffered considerably from shock after the operation, but was never sick, and the wound healed by first intention. He was fed by nutrient enemata until the fourth day, when champagne and milk were given by the mouth. On January 28th he was able to leave his bed, and soon afterwards he was discharged quite well.

Case 2. *Carcinoma of Descending Colon; Colectomy; Recovery.*—B. D., aged 47, was admitted to the London Hospital under the care of my colleague, Dr. F. J. Smith, on January 28th, 1898, suffering with chronic constipation.

History.—Her illness commenced at the beginning of the summer of 1897 with pain in the abdomen and vomiting. The vomiting occurred about two hours after a meal, and the pain was relieved by it. Some months ago she noticed a lump in the left loin. She had passed blood with the stools occasionally for some years, and had resorted to enemata for constipation for two years. Lately tenesmus had existed. She had lost 2 stone in weight within the last six months. She was transferred to the surgical wards on February 6th. Occupying the left loin just below the level of the kidney was a sausage-shaped tumour, distinctly movable from side to side, and evidently situated in the descending colon. No enlargement of the liver existed. The abdomen was somewhat distended. Carcinoma of the descending colon was diagnosed. As a preparation for resection of the bowel a mild purgative (magnesiæ sulph.) was given, with the result that the constipation became absolute, the abdomen distended, and she vomited.

Operation.—February 10th, 1898. Anæsthetic, ether. The tumour was exposed by a longitudinal incision in the left flank, and found to be a constricting carcinoma of the descending colon just below the splenic flexure. The tumour was brought out through the wound, together with some inches of bowel on each side of it, and was stitched to the parietes, the wound being partially closed. An incision was then made into the bowel on the proximal side of the tumour, and a glass tube (Paul's) was tied in. It was not thought advisable to perform resection and suture of the intestine immediately, owing to the fact that the intestines were considerably distended and the patient's condition not favourable for so serious an operation.

February 21st : The portion of the bowel with the tumour protruding from the wound was cut away level with the skin. The incision was so planned that more of the concavity than of the convexity of the protruding knuckle was removed, in order to permit subsequently of easier approximation of the ends. An attempt was made to prevent formation of a spur

by suturing together the mesenteric borders of the ends. Microscopic examination of the tumour showed it to be a typical columnar-celled carcinoma. An interval of about a month was allowed for the inflammatory effusions around the wound to become absorbed.

On March 25th, under ether, an incision through the skin and parietes was made around the colotomy wound, and this was continued above and below the artificial anus for a considerable distance. These incisions were carried down to the peritoneum, which was then separated from the parietes for a distance of $1\frac{1}{2}$ inches to 2 inches all round the bowel. The collar of skin attached to the divided ends of the bowel was removed, and the latter were refreshed, except at the united mesenteric borders. The ends of the bowel were then brought together by two rows of sutures, one uniting the mucous membrane, and the other the peritoneum and muscular coats. The bowel with the peritoneum was then pushed inwards, and the parietes united with silkworm gut, the wound being completely closed with the exception of a small gauze drain. Food by the mouth was given on the fourth day.

On April 3rd, nine days after the operation, a small fistula formed, and a slight fæcal discharge appeared on the dressings. Subsequently the wound was washed out with 1 in 4,000 solution of perchloride of mercury whenever the dressings were changed, but there was no further discharge of fæces. The sinus was quite closed on April 20th. The patient was then allowed to get up. She is now (October, 1899) perfectly well, with no symptom of recurrence, and no enlargement of the liver.

The propriety of draining the perforated intestine at the first operation in Case 1, instead of attempting an immediate resection, is scarcely open to doubt. This remark applies to the present day, when Murphy's button is so much used, nearly as forcibly as it did in 1896, when the case related occurred, and it would apply more strongly to the large intestine than to the small. The introduction of Murphy's button has not in my opinion affected the justice of the conclusion expressed by Mr. Greig Smith as follows: "I have come to the conclusion that to resect living intestine during obstruction is surgically unsound, and I doubt if any improvement in methods will ever make the operation justifiable. The shock and the illness which follow resection of bowel are too great to put on a patient suffering from intestinal obstruction."* It has occurred to me that a perforation or other limited lesion of intestine, not suitable for suture, might be treated by an invagination of a portion of the circumference of the bowel, the invaginated part being retained in position by sutures; but I have not had a favourable opportunity of putting this to the test. Before performing resection I considered whether I would suture or use a Murphy's button,

* 'Abdominal Surgery,' vol. ii, p. 634.

and came to the conclusion that for the large intestine, at any rate, the former was the safer.

In regard to Case 2 the same remarks concerning immediate resection at the first operation apply, although the obstruction was not nearly so acute as in the first case related. An amount of partial obstruction had, however, existed for a considerable period, and this during the day or two preceding the operation had become complete.

I would strongly advocate the method of extraperitoneal suture as the safest, and that which is most free from anxiety. It is, however, not easy to strip up the peritoneum from the parietes to a sufficient extent without tearing it; but slight rents may be closed with a continuous suture of fine silk. The suturing of the intestine is also more difficult, owing to the position of the ends of the intestine. It was especially so in the case reported, because the walls of the bowel were sodden and very friable, and I had at the time considerable doubt as to whether the sutures would hold. They did not, in fact, do so completely, although the ultimate result was not affected.

Mr. W. H. Battle said there was a general agreement amongst surgeons as to the inadvisability of resecting while there was actual obstruction. With respect to the method of treatment in cases of malignant growth where obstruction was present, he pointed out that it was not always possible to get the large intestine up to the surface. In one case under his care, for instance, where there was a tumour in the splenic region, the abdomen was so distended that it could not be got within 2 inches of the skin, and he was obliged to do a colotomy above the seat of obstruction, and to insert a Paul's tube. Five weeks later, he excised the growth and artificial anus, and did a lateral anastomosis by suture. He had shown that patient at the Harveian Society, and she had since continued perfectly well. In another case he had commenced by the method described by the author, bringing the growth outside and putting in a Paul's tube, but, unfortunately, the patient did not rally from the operation, dying, three days later, from the effects of long continued obstruction. He observed that at St. Thomas's Hospital their experience of excision for growths was in favour of lateral anastomosis and the use of sutures without apparatus. He had done one resection of intestine for growth at a time when the symptoms were slight and the case was apparently favourable for the operation; he used Murphy's button, but the patient died a few days later with localised peritonitis. He pointed out that it was extremely difficult to control the amount of pressure used in closing the button, and when once closed it could not be undone. In a similar case in future he would prefer lateral anastomosis by suture. In another case shown with the first mentioned at the Harveian Society, the patient was a woman suffering from chronic intestinal obstruction, due to a growth in the sigmoid flexure. He had

nad recourse to Paul's method, but afterwards had found it very difficult to re-establish the continuity of the canal, the septum formed having become very rigid. He ultimately made another incision through the abdominal wall, and performed lateral anastomosis between the two portions of intestine, leaving part of the septum practically untouched, and then he passed a strong silk ligature round it and closed the artificial anus. The patient was, at present, apparently in good health.

Mr. Wallis agreed in deprecating the use of a button in operations on the large intestine, his own experience not having given good results, probably because it was extremely difficult to appreciate how much pressure was necessary to fix the button, a certain amount being required. On the other hand the very fact of the large intestine being so thick was a suggestion that ordinary sewing was really most effectual, for there was plenty of material to work upon, and they could put in sutures with much more confidence than was the case in the small intestine.

Mr. Eve, in reply, said that with regard to the formation of a spur he himself endeavoured in his last case to avoid it by removing more of the concavity than of the convexity.

May 15th, 1899.

THE ANNUAL ORATION—"SHAKESPEARE AND THE MEDICAL SOCIETY."*

By Alban Doran, F.R.C.S.

Mr. President and Gentlemen,—"Present fears are less than horrible imaginings," so that I feel a sense of relief now that I am really about to deliver this Oration. When the Council informed me last summer that I was selected Orator I felt "most admir'd disorder," like Macbeth's guests. I held then the office of an Ariel to the Society, being "correspondent to command" to foreign parts, and I had just heard curses both loud and deep follow my proposal that the indexing of our valuable 'Transactions,' which I have for long maintained to be desirable, should be undertaken—but by somebody else. "Something too much of this," however, as Hamlet said to Horatio. I was selected. I thank the President, Council, and Society for the honour which they have conferred upon me, and, like those humble people, the players in *A Midsummer Night's Dream*, my "true intent is all for your delight."

* The quotations throughout this address are taken from Dyce's Text.

The choice of a subject rendered me "perplex'd in the extreme," like Othello. Still it is right that I should dwell upon it. "Little shall I grace my cause in speaking for myself," but as Ulysses says in *Troilus and Cressida*, "One touch of Nature makes the whole world kin." Most of you have had or will have to choose a theme for an address. At first I thought of "Medicine: a Noble Profession," but, like Troilus, "I cannot fight upon this argument, it is too starv'd a subject for my sword." Lectures on this lofty theme are usually "full of wise saws and modern instances." I will follow Richard the Third's advice to Queen Elizabeth of York when she alluded to the asphyxiating of the princes in the Tower, and harp not on that string. As to questions where I might speak after some experience, they set me thinking of statistics of abdominal operations, till I felt, like Lewis in *King John*, that to you they would be "as tedious as a twice-told tale vexing the dull ear of a drowsy man." Some of you may say, like Francisco in *Hamlet*, "For this relief much thanks." I soon put aside all thoughts of deciduoma malignum as my subject; had I selected it you would have found me "tedious," like Polonius, Dogberry, and Verges.

At length I bethought me of the Bard of Avon. His observations upon human nature can be fitted to anything human in any age, collectively or individually. So I decided to apply his wisdom to the ways and doings of our Society and our profession. It happens, as you all know, that our President opened this 126th session with some instructive remarks on a precious MS. work in our library, the diary of a Vicar of Stratford-on-Avon,* who lived there not long after Shakespeare's death and recorded certain facts and traditions about the Bard. Allow me, then, to conclude the session with a little more about him. I expressed my intent to our President, as the conspirators communicated their plot to Brutus, for "that which would appear offence in us, his countenance, like richest alchemy, will change to virtue and to worthiness." Our President, like the King in *Hamlet*, wished last autumn to know something about the "argument," lest there should be any "offence in't." I replied that I had not even begun the "argument," but that there would be "no offence i' the world," and thus I won his approval.

* *See* p. 3. Mr. Sidney Lee's 'Life of William Shakespeare' throws much light on the Bard's later associations with Stratford.

In awarding due praise to Dr. Bucknill, Dr. John Moyes, of Largs, and others who have written systematic works on Shakespeare and medicine, I must add that their method is not suited for the present oration, nor need I discuss their subject in a general sense, as has been done by Mr. Lemuel Griffiths, of Clifton, in an excellent address published in the fifth volume of the 'Bristol Medico-Chirurgical Journal.' Nor, on the other hand, need I weary you with a string of Shakespearian samples laboriously extracted from Bartlett's 'Familiar Quotations,' or from Cowden Clarke's 'Concordance,' or from some so-called "beauties" of Shakespeare. Let us see how far the poet's thoughts suit our case.

This Society, as a society, encourages us to speak, and "conference," says Bacon, "maketh a ready man." We are more or less obliged to talk about what we understand, and the circumstances of a meeting are not such as make men timid. Hence in our discussions we see none of that painful nervousness shown by the student at an oral examination, or by the dinner guest unexpectedly called on to reply to a toast. The victim feels like banished Norfolk in *Richard the Second*:—

> And now my tongue's use is to me no more
> Than an unstringèd viol or a harp,
> Or like a cunning instrument cas'd up,
> Or, being open, put into his hands
> That knows no touch to tune the harmony,
> Within my mouth you have engaol'd my tongue,
> Doubly portcullis'd with my teeth and lips.

On the contrary, in a Society like ours, though high oratory is perhaps not conspicuous, many of us speak trippingly on the tongue, and few, if any of us, saw the air with our hands, but we use all gently. In discussion we appear to acquire and beget a temperance that may give it smoothness. Let us trust that we rarely, if ever, speak more than is set down for us by the limits of our subject, a defect less to be excused than "gagging," its homologue on the stage, which Hamlet, whose language I am borrowing, condemns as villainous, showing a most pitiful ambition in the fool that uses it; nor, as the Duke in *Measure for Measure* would say, do we think the man of safe discretion that does affect it.

Let us consider ourselves first as doctors, for of such are the

Fellows of this Society. Shakespeare puts us forward both as authorities and breadwinners. There are unqualified practitioners in his plays; thus Richard II, when trying to reduce Bolingbroke and Norfolk's mental temperature, says, "this we prescribe though no physician." The black sheep, the Calibans of our profession, are not of our fold; we leave the Prosperos of the Colleges and the General Medical Council to deal with them. We encourage these worshipful authorities, as the host of the Garter encouraged Falstaff about his followers: "discard, bully Hercules; cashier: let them wag; trot, trot." Of a certain odious professional crime, we may say, as Stephano says of a misdeed of Caliban's, "There is not only disgrace and dishonour in that . . . . but an infinite loss."

The doctor in Shakespeare's plays is placed favourably before the audience. Jokes at the expense of medical men teem in French and English comedies of a later age. Molière was right in condemning the medical education of his time, and in reminding us that patients want to be cured.* Thirst for fees is never made an attribute of the doctor in Shakespeare's plays. Macbeth's physician alludes to profit, but in respect to a shady patient, and when fearing personal danger. "Were I from Dunsinane away and clear, profit again should hardly draw me here." In the generation after Shakespeare, according to the researches of the late Dr. Creswell Rich of Liverpool, there were those who considered that whilst, of course, we must practise, we are no longer "doctors" when we cure for money. A medical bard of the days of the Commonwealth embodied this idea in a clever rhyme†:—

> *Doctors* or *Teachers* they of *Physick* are
> (Whether by Pen they do it or in Chair
> With lively Voyce), that teach the way to know
> *Man's Nature*, *Health* and *Sickness*, and do show
> *Diseases*, *Cause* and *Cure*. But they who spend
> Their Life in *Visits* and whose Labors end
> In taking *Fees* and giving Paper-scrowls
> Factors of Physick are; and none but *Owls*
> Do count such *Doctors* that *no Latin* know
> From which that Name did to our Language flow.

* "Ce qu'il y a de fâcheux auprès des grands, c'est que, quand ils viennent à être malades, ils veulent absolument que leurs médecins les guérissent," *Le Malade Imaginaire*, Act ii, Scene 6.

† 'British Medical Journal,' vol. ii, 1883, p. 1103. Arthur Creswell Rich,

This epigram appears at the end of the preface in a copy of Riverius's 'Practice of Physick,' and is signed "W. R. Doctor, and Factor of Physick, from my study at Montpelier, July 1st, 1653." Most of us in these days are like this forgotten Dr. W. R., and the distinction which he applies to himself is quite reasonable and inoffensive.

I cannot avoid dwelling awhile on the two noble doctors in *Macbeth* and *King Lear*, though many have already spoken of them in medical addresses. One delivers a speech among the most beautiful in the bard's plays, and that is saying a good deal.

For some reason Lear's medical attendant is styled "a physician," Macbeth's "a doctor of physic." Lear's is made the more successful of the two. Of course there is no question of history in *King Lear*. In the days of British kings there was no such thing as a King of France, and absolutely no political homologue to a Duke of Burgundy. Nor did Shakespeare create this physician to typify the medicine man of ancient Celtic Europe. Such a task was beyond his knowledge and power even if suited for dramatic treatment. Shakespeare clearly makes Lear's physician like to a doctor of his own times. It seems to me that the reason why Lear's doctor is made better than Macbeth's is purely dramatic, and not a direct way, independent of the plot, of demonstrating that one doctor of repute may be more skilled than another in closing the eye of anguish and ministering to a mind diseased. Shakespeare felt that we want to see the deeply-wronged Lear kindly treated, whilst dramatic morality demands that Macbeth's doctor should remind the usurper that our profession cannot cure guilty consciences.

Cordelia consults the physician about her sire's delirium:—

> What can man's wisdom
> In the restoring his bereavèd sense?
> He that helps him take all my outward worth.

She did not run her court physician on club principles. He was

M.B. Lond., died in May, 1888, and an obituary notice will be found in the 'Journal,' vol. i, 1888, p. 1091. I do not know what has become of the copy of Riverius's book which contains this remarkable epigram. I suspect that Dr. Rich modified the spelling, which (except "voyce" and "scrowls") seems too modern for 1653.

worthy of his hire in her generous opinion. His reply is a fine speech :—

> There is means, Madam :
> Our foster-nurse of Nature is repose,
> The which he lacks ; that to provoke in him,
> Are many simples operative, whose power
> Will close the eye of anguish.

Like one of our past Presidents, Mr. Knowsley Thornton, he believed in narcotics in treatment. Though medicinal herbs were called "simples," the mixtures made up from them were highly complicated. Still less can we apply such a term to the narcotics and hypnotics of to-day. I have closed the eye of anguish with diethyl-sulfone-methyl-ethyl-methane, and find it sometimes superior to dimethyl-methane-diethyl-sulphone. Trional and sulphonal certainly sound better. A more serious matter in respect to the very unsimple nature of modern chemical compounds is that multiplicity of synonyms just indicated. We hear of inquiries as to trying the effects of a little analgesine or even some phenazone on a patient who cannot tolerate antipyrin.

Lear's physician gets his patient to sleep. Afterwards he wakes the King so that the old man may see that his beloved, misunderstood daughter is tending him. The doctor prescribes music, preferring it apparently to brandy and milk, or a new patent food. Anyhow, the rest has restored Lear's faculties. "I am a very foolish fond old man, four score and upward, not an hour more nor less," says the King. The doctor has succeeded.

Macbeth's medical adviser has a patient who has incited her husband to murder his guest. She is supposed to be sinking, just like "the grand conspirator, Abbot of Westminster" in *Richard the Second*, from "clog of conscience and sour melancholy." The doctor deliberately tells Macbeth that his wife is "not so sick . . . . as she is troubled with thick-coming fancies, that keep her from her rest." The usurper replies, "Cure her of that," and makes a well-known speech. Several purely medical critics consider that the doctor is meant to show want of tact when he says "Therein the patient must minister to himself." I believe, however, that a purely dramatic and moral idea runs through this consultation. There must be no help for the wicked—nothing but care, ruin, and death for the guilty couple. Just as the doctor, among other characters, must comfort Lear, who deserved to be comforted, so he must fail to comfort Macbeth and his wife,

who had forfeited the sympathies of humanity. From our point of view no doubt the doctor was unwise in saying that "therein the patient must minister to himself." Such a remark offends people, and it is natural that Macbeth should reply, "Throw physic to the dogs!" We know how a patient ministers to himself, in respect to so material an act as making his own poultice. But I believe that this was not Shakespeare's idea. He meant that the doctor, like the usurper's officers and men, could not ward off the pangs of conscience and the coming ruin of the King and Queen. The apparently tactless remark was meant to be conscience speaking through the doctor's mouth, and conscience is not supposed to possess the tact of a Court physician.

Later remarks of Macbeth to his physician simply imply desperation. If he could cure Scotland by physic, the usurper would applaud him to the very echo that should applaud again. But Macbeth was not criticising physic; he knew that his country could only be cured by being purged of himself, its real disease.

Let us mark that Timon of Athens, another desperate man, abuses doctors when he says to the thieves:—

> Yet thanks I must you con,
> That you are thieves profess'd; that you work not
> In holier shapes; for there is boundless theft
> In limited professions . . . .
> . . . . Trust not the physician;
> His antidotes are poison, and he slays
> More than you rob.

No reader of Shakespeare thinks that our profession is here attacked. The passage is meant to show the blindness of judgment bred by disappointment. The use of the doctor for dramatic purposes is evident here and nearly, though not quite, as evident in *Macbeth*. In neither is there any satire directed against medicine.

There is also an English doctor in *Macbeth*. In a passage which always puts me in mind of our clinical evenings he relates to the Scottish exiles how Edward the Confessor cures the "king's evil":—

> There are a crew of wretched souls
> That stay his cure; their malady convinces
> The great assay of art; but, at his touch
> Such sanctity has heaven given his hand,
> They presently amend.

Prince Malcolm adds :—

> . . . . Strangely-visited people,
> All swoln and ulcerous, pitiful to the eye,
> The mere despair of surgery, he cures.

Here the Bard is really glorifying Edward the Confessor, the last Englishman by family who ever sat on the throne. Then follows a remark which simply means that the rulers of England in Shakespeare's time clearly had sanction from Heaven as well as from earth :—

> . . . . Tis spoken,
> To the succeeding royalty he leaves
> The healing benediction.

The doctor is made a prophet to play up to the loyalty of an audience already loyal to "Eliza and our James," and to make those sovereigns figure as Heaven-sent successors to a national hero whose memory was deeply revered.

Shakespeare never meant to hold up this English doctor to ridicule because he admitted that scrofula was the mere (complete) despair of surgery. It remained so till very recent days, and some of its forms still "convince the great assay of art." Cancer and certain other diseases still defy us. Let us study them all the more earnestly. Like the watchman in *Romeo and Juliet*, "we see the ground whereon these woes do lie; but the true ground of all these piteous woes we cannot without circumstance descry." We much watch hard cases, and the Medical Society does so. "Strangely visited people . . . . pitiful to the eye," are what we collect for our clinical evenings. Our aim is that they should be no longer pitiful to the eye, and that their visitation should not remain strange, but be made easy to understand and overcome. Do we succeed? We can say, Yes! Let us look at the records of the Society 20 years ago. Cases were written about which nobody would bring forward now, for we all understand much more about them. Sufficient information is to be found in our textbooks.

Cornelius, the physician in *Cymbeline*, is placed in an entirely favourable light. But just as the Bard did not create the two doctors in *Macbeth* in order to satirise medicine, so he did not mean Cornelius to glorify our profession. The character is part of the dramatic machinery devised to protect Imogen against her enemies. Dr. Butts, an historical personage, is not represented

in his professional capacity in *Henry VIII.* Dr. Caius is held up to ridicule, but as a comic foreigner, an elderly wooer, not as a doctor.

In *All's Well that Ends Well* the Court physicians fail to cure the King of France's thoracic fistula, but the plot turns on the success of the heroine who uses a remedy introduced by a deceased doctor—her own father. It would be interesting to know how the fistula was closed. Helena's "receipt" seemed, according to Boccaccio, to be purely herbal (*polvere di certe erbe utile a quella infermità*),* so that there was no drainage, still less excision of a rib. In the original story† we are distinctly told that the fistula (*fistola*) was thoracic (*nel petto*).

Shakespeare's attitude to surgery now claims our attention. The surgeon held a very low social position in the sixteenth century. He is sent for in *Romeo and Juliet* and in *Othello.* In *Twelfth Night* Sir Toby Belch says: "Sot, did'st see Dick surgeon, sot?" Nowhere is the surgeon exalted, yet there are numerous allusions to surgery, and much of the general wisdom in Shakespeare's plays especially applies to surgery. Often is the surgeon, like the captive Richard II, a sworn brother to grim necessity. We must be prompt when surgical interference is clearly indicated. "Our doubts are traitors, and make us lose the good we oft might win by fearing to attempt," as Lucio says in *Measure for Measure.* "Present" or immediate surgical assistance must often

* Boccaccio, 'Decameron,' giorn. iii, Nov. 9, Martinelli's edition, 1762.

† Boccaccio, *loc. cit.* Dr. Bucknill, in 'The Medical Knowledge of Shakespeare,' quotes the passage, which shows that the Italian author spoke of a thoracic fistula. Dr. Moyes ('Medicine and Kindred Arts in the Plays of Shakespeare') traces Shakespeare's version to an intermediate tale by Painter, where it is distinctly stated that "the French King had a swelling *on his breast*, which by reason of ill-cure was grown to be a *fistula*." I have added above Boccaccio's specification that the receipt was a powder of certain herbs. From what I can make out, the King was probably the Dauphin Charles, who was a youth of 16 in 1353, when the 'Decameron' was published. When a child he had a long illness, and an abscess discharged under his arm. I find that his chronicler, Christine de Pisan, states that Charles remained sickly and subject to feverish attacks all his life. All sorts of nostrums were tried, it appears. In 1380, when he was Charles V of France, the fistula ceased to discharge, and he died within a fortnight. Boccaccio may refer to an earlier case, but there was probably much talk when he wrote the story about the Dauphin's illness, so that he chose thoracic fistula as the particular disease which Helena managed to cure.

be administered, like "present medicine" in King John's case, "or overthrow incurable ensues." Like Richard III, we have heard that "fearful (that is, timid) commenting is leaden servitor to dull delay," which is dangerous when we have hæmorrhage or strangulated hernia to deal with. On the other hand, the surgeon must not trust too much in novelties. They soon come to his hearing, as in the case of Richard II; for York asks John of Gaunt: "Where doth the world thrust forth a vanity (so it be new, there's no respect how vile) that is not quickly buzz'd into his ears?" Too many novelties prove to be devoid of the merit of originality. What seems new is too often assumed to be new without scrutiny, so deeply does any apparent innovation fascinate us. "All, with one consent," says Ulysses, "praise new born gawds, though they are made and moulded of things past."

We must not forget that the fame of a surgeon may sanction a novelty on insufficient grounds. An able operator is naturally admired. His followers imitate him in the operating theatre, where he is probably their superior. They usually go a step further and support his practical work, on the ground of scientific theories which have never entered his head. He is in the position of Antonio and Sebastian in the *Tempest*, when they say, in respect to their conspiracy:—

> For all the rest,
> They'll take suggestion as a cat laps milk,
> They'll tell the clock to any business that
> We say befits the hour.

Hence, when the "business" is the drying of the peritoneum with sponges, his disciples find that sponging is the best means of avoiding sepsis. When the drainage tube is said by the great operator to "befit the hour," forthwith that appliance is found, on scientific evidence, to be an absolute necessity as a guard against deadly germs. Then comes a gentleman who practises flushing of the peritoneum with saline solution. At once experimenters demonstrate that the drainage tube is a dangerous conveyor of sepsis, and that it is the bounden duty of every surgeon to reject it and adopt the new practice. Those who will not take suggestion as a cat laps milk are "but as the cuckoo is in June, heard, not regarded," as Henry IV said when he scolded his son.

A surgical Ulysses is justified in being shy about enthusiasm

for new operations, especially when he knows that the knife can be dispensed with. He rightly condemns those who "tax our policy and call it cowardice; count wisdom as no member of the war; forestall prescience and esteem no act but that of *hand*." Novel operative measures as reported too often lack after-histories. "Striving to better, oft we mar what's well," says Albany; and when his wife protests, he adds, "Well, well, the event!" In Goneril's case "the event" or after-history was disastrous, but Shakespeare did not omit to include it in his report.

In the recognised fields of operative surgery, however, we cannot have too much courage, experience, and practice. Surgical cowardice is far worse than overzeal for surgical innovations. While we must not let our surgical instincts induce our reason to argue in favour of operating when we are not sure that the case may not do without it, we at the same time must never allow timidity to urge us to refrain from the knife when we know that it offers the patient the best chance of recovery. We must be specially brave and conscientious in this respect when the case is specially dangerous, otherwise a fatal termination, should we decline to operate, may be a greater opprobrium than death following an operation which we know to be our duty. As Greig Smith most righteously taught, we must try to save other people's lives even if we ruin our own statistics, otherwise those statistics will come under Touchstone's "lie circumstantial." "Of all the wonders that I yet have heard, it seems to me most strange that men should fear," said Cæsar. This certainly should apply to operating. Custom will make it in us a property of easiness, till we become less and less like the gravedigger in *Hamlet*, to whom Horatio applied this expression.

These observations on surgery refer to the surgeon in abstract. They demonstrate sources of error ineradicable from human nature. There can be no individual surgeon of experience who has never placed in a new operation or in the teaching of others confidence which has proved to be ill-founded, and who has never wished that an operation which he has been obliged to perform under desperate circumstances had not fallen to his lot. "Who calls out on pride that can therein tax any private party?" says Jacques. As with satires on pride, so it is with criticism on the inherent weaknesses of man as a surgeon. Such criticism

"taxes," that is to say, condemns, no "private party," no individual surgeon. It concerns us all, and so we must never cease to strive alike against the abuse of our zeal and the promptings of our fear.

Let us turn now to our library. Prospero said: "My library was dukedom large enough," and certainly Dr. Allchin, our retiring librarian, has ruled his duchy well. Like Shakespeare, we do not rely too much on books. Iago complains that Cassio only knows the bookish theoric, mere prattle without practice. Biron, in *Love's Labour's Lost*, carefully distinguishes between study and books:

> Study is like the heaven's glorious sun,
> That will not be deep search'd with saucy looks ;
> Small have continual plodders ever won,
> Save base authority from others' books.

We certainly all agree with Biron. Compared with personal observation—

> Other slow arts entirely keep the brain ;
> And therefore, finding barren practisers,
> Scarce show a harvest of their heavy toil.

We go further than Biron meant—we study men as well as women. Our fellow-creatures are the books, the arts, the *academes* that show, contain, and nourish all the world. We also investigate morbid structures and *post-mortem* appearances. As Northumberland says on hearing the rumour of Bolingbroke's return: "Even through the hollow eyes of death I spy life peering," an expression which recalls to our minds "*Nec silet mors*," the beautiful motto of a sister society, the Pathological. Books are a means, not an end. Yet means are wanted, and we have a fine stock. A good clinical report is like a good picture, "it tutors Nature ; artificial strife lives in these touches, livelier than life," as the poet in *Timon of Athens* remarks. A certain amount of experience renders authorship almost a duty. "The fire i' the flint shows not till it be struck," and the experience of an earnest worker may never show beyond the limits of his practice or his hospital till it is committed to paper.

This duty of making public all useful medical knowledge leads us to think of the most prominent features of our Society—our papers and our discussions. We all feel as Ulysses did when he upbraided Achilles for not coming to the meetings of the

Grecian heroes, where the discussions, we must admit, were of more general interest than ours:

> No man is the lord of anything
> (Though in and of him there be much consisting)
> Till he communicate his parts to others;
> Nor doth he of himself know them for aught
> Till he behold them form'd in the applause
> Where they're extended.

Most assuredly no man can feel that his knowledge and experience are of any definite value for the relief of disease or for the advancement of science until he communicates them to others, as he can do at our meetings, and sees what reception or "applause" is accorded to them here where they are made public or "extended." Good papers are based on observation and experience, "set in a notebook, learn'd and conn'd by rote," like Cassius's faults or a neurotic patient's account of his own symptoms. As Antonio says in *The Two Gentlemen of Verona* (one of whom was not a gentleman), "Experience is by industry achieved and perfected by the swift course of time." Once that experience is gained, the writing of a paper becomes easy, for we write best about what we know best, and therefore like best. "No profit grows where is no pleasure ta'en," says Tranio in *The Taming of the Shrew*, and "to business that we love we rise betime and go to't with delight," like Mark Antony. The great advantage of a society like ours is, as I have already observed, that it obliges us to confine our observations to matters about which we have some knowledge and experience. Never, I trust, do we forget that we should all listen a great deal more than we speak and write, so that we may profit by the collective knowledge and experience of the multitude of counsellors in the Society. Like Gloster, our younger colleagues must not be easily won to our requests for papers. Nevertheless, they too should communicate their parts to others after they have learnt here how to do it. Parolles, in *All's Well that Ends Well*, says to Helena when she defends spinsterhood, that to speak on the part of single life "is to accuse your mothers, which is most infallible disobedience." On the same principle, for our junior brethren to speak in favour of abstention from papers, and to defer indefinitely their maiden speeches is to accuse their elders, which we cannot but think "most infallible disobedience." Of a junior amongst us we may say, as was said of Proteus's proposed travels, that it "would be

great impeachment to his age in having"—read no papers—"in his youth."

A good paper cannot always be free from justifiable criticism, which none the less may offend those of an opposite opinion. Yet Goneril spoke truth when she said: "All's not offence that indiscretion finds and dotage terms so." We all, even if still young, tend to dote on our own conclusions and theories, and never like to find them supplanted, but they must in their turn give way to others, just as they once supplanted earlier teaching. Even an apologetic sentence to this effect, in a new paper, only irritates us, just as the King deeply offended Hamlet by reminding him that if he had lost his father, "that father lost, lost his." As Mephistopheles says to the student in Goethe's *Faust:*

> Grau, theurer Freund, ist alle Theorie
> Und grün des Lebens goldner Baum.

In truth every theory is born old and grey, and life is ever young and green. Let us not think that our own theories are immortal, but let us rather imitate the modesty of Dr. Samuel West, who concluded his third Lettsomian Lecture with the following words: "On a subject so full of difficult and contentious questions I cannot expect that my views will commend themselves to all. Yet defined opinions challenge criticism, and criticism tends to advance knowledge."* We all know how well Dr. West, like Buckingham, left nothing fitting for his purpose untouched or slightly handled in debate.

In reference to another, our 'Transactions' show that we always "speak brotherly of him," not in the spirit of Oliver, who wanted his brother Orlando's neck to be broken. The truth we speak never seems to lack gentleness, nor time to speak it in; in short, we are more polite than my Lord Sebastian. Nevertheless, we may chide away a friend by opening his eyes to an error, just as Prince Arthur chid away a friend when, on the contrary, his eyes were in danger of being put out.

Closely associated, both in a literary and a scientific sense, with the preparation of papers is the question how far we can rely on foreign authorities. We can understand our own teachers, and we know how much weight can be attached to the opinion of a living British authority as we can easily become acquainted

* *See* p. 192.

with the man, we can judge of him by his appointments, and he communicates his ideas in our own tongue. The same is not the case with the author of a foreign monograph or clinical sketch, the value of which it is important to estimate before we quote it. Foreign opinions should never be taken from a translation in abstract. We must translate the original, or induce a friend who knows the language to translate it for us. In short, owing to the excellent work done abroad, "the tongues," as Shakespeare would say, are indispensable. Don Pedro, in *Much Ado About Nothing*, assures Beatrice that Benedick "hath the tongues," an accomplishment socially advantageous to a Renaissance gallant, but a necessity to us who write papers for the Society. Our last volume shows that we own our indebtedness to the foreigner. It is indeed meet that we should do so. The Fellows of our Society are not in the habit of quoting without acknowledgment. Let us remember a significant scene in *The Two Gentlemen of Verona*. Valentine enters a forest "on the frontiers of Mantua," where "certain outlaws" make him stand and deliver. He expresses a desire to join their band. One outlaw says, "Have you the tongues?" Valentine's reply being in the affirmative, another robber exclaims enthusiastically, "This fellow were a king for our wild faction." We know only too well that "certain outlaws" of literature, known as plagiarists, make free use of foreign authors without being too ready to acknowledge them. But I do not think that any Fellows of our Society belong to that "wild faction."

There is another advantage in a Society like the Medical. It teaches us command and management. We have a President and Council to govern us, for—

> The heavens themselves, the planets and this centre,
> Observe degree, priority, and place,
> Insisture, course, proportion, season, form,
> Office and custom, in all line of order.

"What with our Council we have done," as Richard II says, we all know.

Sometimes a specially distinguished gentleman obliges us by ascending our rostrum. This session was thus favoured by Sir William Broadbent.* We may assure him, as the Lords assured

* "The Conduct of the Heart in the Face of Difficulties" (*see* p. 78).

the King of France in *All's Well that Ends Well*, that "our hearts receive your warnings." But Gloster's heart in *King Lear* conducted itself ill in the face of difficulties.

> His flaw'd heart—
> Alack ; too weak the conflict to support !—
> Twixt two extremes of passion—joy and grief
> Burst smilingly.

Yet Loison's recent statistics of wounds of the heart* show that in prose, if not in poetry, that organ stands a great deal, and will even allow needles to be thrust into it and then pulled out by the surgeon. Herein Fortune, as Antonio would say, shows herself more kind than is her custom.

In conclusion, if we ever feel momentary doubts about the real value of our Society, and if others ask what "good" is to be got out of papers and discussions, it must be remembered that such a sentiment is unwholesome and akin to the thought that all medical research and progress is an illusion. It is a sentiment pardonable in Macbeth and Timon of Athens when driven to desperation, but unworthy of sober-minded doctors. "Contempt," says Carlyle, "is a dangerous element to sport in; a deadly one if we habitually live in it." What though there seem but little in any one paper or discussion, what if even the new method for healing the sick which it advocates should prove less satisfactory on further trial than its author expected? What if a weighty scientific monograph show but little, considering the time and labour spent on its preparation? The same objection applies to the clinical and surgical labour and to the microscopic and laboratory work which these productions faithfully register. The same objection has been raised in regard to all great efforts. As Agamemnon says :

> The ample proposition that hope makes
> In all designs begun on earth below
> Fails in the promised largeness. . . . . Why, then
> Do you with cheeks abash'd behold our works ;
> And call them shames, which are, indeed, naught else
> But the protractive trials of great Jove
> To find persistive constancy in men ?

"In the reproof of chance," adds Nestor, "lies the true proof of men." "Wise men," says Queen Margaret of Lancaster,

* "Des Blessures du Péricarde et du Cœur," 'Revue de Chirurgie,' January, February, June, and July, 1899.

"ne'er sit and wail their loss, but cheerly seek how to redress their harms." If a colleague can show us better results and demonstrate any scientific error we have made, let us thank him and cheerily seek the sources of the fallacies which have misled us. Then it shall go hard, as Shylock would say, but we will better the instruction.

After all, I am sure that it is seldom that we feel doubts about the value of our work. Papers and discussions make us articulate, as Carlyle would say, our yet more important researches so beneficial to science and humanity. Matters of detail may seem tedious, but "most poor matters point to rich ends," as Ferdinand in the *Tempest* was bound to admit. We feel, when labouring for our liege lady Medicine, as he felt when working for his Miranda. For my own part, "the mistress which I serve quickens what's dead, and makes my labours pleasures."

We who make up the Medical Society are members of an essentially active profession, and while we remain in our prime let us do our best here actively. For age, like infancy, is mostly passive, or, as Edgar reminds us in *King Lear:*

> Men must endure
> Their going hence, even as their coming hither;
> Ripeness is all.

CLINICAL EVENINGS.

November 14th, 1898.

CASE OF GASTRO-ENTEROSTOMY PERFORMED FOR INOPERABLE DISEASE OF THE PYLORUS.

By the President, Edmund Owen, F.R.C.S.

The patient, a man, aged 31 years, for a twelvemonth had discomfort in the pyloric region, with vomiting after meals. He had lost his appetite, and could neither work nor sleep; he was greatly emaciated. The stomach was much dilated. On July 18th abdominal section was performed; the pylorus was obscured and fixed by a hard mass surrounded by enlarged lymphatic glands. A communication was made between the front of the stomach and an adjacent piece of jejunum by means of a Murphy's button. The man could now eat and drink anything; he had lost his dyspepsia and had become almost robust. Nothing had yet been seen of the button, but, wherever it was, it was doing no harm.

Mr. Battle asked what size of button was used, also the exact portion of the stomach with which the communication was made? He also asked how soon after operation food was given by the mouth, and as to the duration of the previous illness? He inquired what was the amount of the glandular enlargement? He gathered that the President believed it to be a case of malignant disease of the pylorus, in which case, of course, the prognosis was necessarily unsatisfactory. He himself had last year operated on a man, aged 28, for pyloric obstruction of six weeks' duration, though the symptoms were acute. Although the duration of the malady was apparently but short, he found extensive growths in the glands and multiple growths on the peritoneum. The patient did extremely well after the operation, but died six weeks later from malignant disease of the pleura.

The President, in reply, said the lumen of the stalk of the button was about the size of the top of his little finger, and when it sloughed away it would leave an aperture about the size of a shilling which he feared might go on contracting. The disease in this case had lasted about 12 months. He made an opening into the stomach in the anterior part,

low down, quite clear of any vessels. He had fixed a piece of jejunum to the front of the stomach where it was in apposition. The question had been raised as to the advisability of making the opening in the anterior part of the stomach on account of the risk of a kink, but his experience did not suggest any difficulty of this nature. He began to give the patient home-made beef jelly on the day after the operation. He thought it was a malignant growth, and the prognosis was, therefore, decidedly bad.

CASE OF VASCULAR MURMUR OF OBSCURE ORIGIN, AUDIBLE AT THE BASE OF THE RIGHT LUNG.

By Hector Mackenzie, M.D., F.R.C.P.

The patient was a man, aged 41 years, who had been under treatment for recurrent hæmoptysis at the Brompton Hospital during the last four years. Throughout the first period of attendance, from August, 1894, to May, 1895, there were five attacks of hæmoptysis, which were all slight, and the only abnormal physical signs then observed were diminished resonance, bronchial breathing, and pectoriloquy at the right apex. The patient kept fairly well until August, 1897, when hæmoptysis recurred, and he again presented himself at the hospital. He had a cough with yellow expectoration, and complained of night sweats and loss of appetite. The physical signs at the right apex were the same as before, with the addition of crepitations, but it was now discovered that at the base of the right lung a very peculiar vascular murmur was present. The murmur was loud and blowing, and occupied nearly the whole period of the cardiac cycle. In character it very closely resembled the murmur produced by a communication between an artery and a vein, such as sometimes occurred in cases of aneurysm. The murmur increased in intensity with inspiration, but was not obviously altered when the patient held his breath either in inspiration or expiration. The position of maximum intensity was situated over the seventh, eighth, and ninth ribs in the posterior axillary line, on either side of which the murmur was distinctly audible for 2 or 3 inches. There were no other abnormal physical signs at the right base, no dulness, no pulsation, no alteration of breath or voice sounds. Since it was first noticed the murmur had not altered either in character or situation. During the last year there had been

numerous slight attacks of hæmoptysis, but during the intervals the patient had been fairly well and able to continue his work as a draughtsman. There had been no change in the physical signs at the right apex. Dr. Mackenzie was unable to offer any explanation of the cause of the vascular murmur. It was possible that it might be due to a vascular growth in the lung. It was unlikely from the situation that it was produced by an aneurysm. The fact that the patient had pulmonary tubercle was sufficient explanation of the attacks of hæmoptysis, which probably were quite unconnected with the condition that gave rise to the murmur.

Dr. Maguire said the only condition he could suggest as the cause of the murmur was an angeioma of the liver. The murmur resembled that heard over the jugular vein in anæmia and, like it, was increased in intensity during inspiration. Moreover, the upper part of the liver seemed to palpation to be somewhat irregular, rising towards the vertebral column instead of sinking. He admitted that angeiomata of the liver were usually small and most frequently embedded in the substance of the organ, but they did occur on the surface, and he had seen one as large as a duck's egg, which would suffice to cause such a murmur.

The President asked whether there were any "mother's marks" which might suggest the murmur being due to a nævoid formation?

Dr. Mackenzie replied in the negative.

Dr. H. A. Caley suggested that the murmur might be generated in connection with the vena cava at its passage through the diaphragm. It was remarkable what a difference there was during inspiration, and this, he thought, was consistent with a relative stenosis of the vena cava in that situation.

Dr. Mackenzie, in reply, said he had not been able to make out any irregularity of the upper border of the liver as stated by Dr. Maguire.

CASE OF ANEURYSM OF THE RIGHT COMMON CAROTID ARTERY.

By Seymour Taylor, M.D.

The patient, a carman, aged 35, had a pulsating tumour about the size of a turkey's egg, in the course of the right common carotid artery. The tumour extended from immediately above the sterno-clavicular articulation up to the level of the hyoid bone. The sternal head of the sterno-mastoid muscle appeared to be stretched over, and by, the aneurysm. No pain or distress was caused by the tumour, and, although the voice was somewhat altered in character, the vocal cords were not affected. All the

symptoms dated from June, 1897, when the man was an in-patient of Westminster Hospital, where both superficial femoral arteries were ligated by Mr. Spencer for aneurysm in each popliteal space. There were no signs of aortic aneurysm, or of aneurysm of any other large artery.

The PRESIDENT observed that if one asked a student the causation of arteritis, he would certainly incriminate syphilis ; but personally he was not so sure that this was an important factor in the etiology of arteritis. He recalled the observation of a surgeon-major in the Indian Medical Service, who commented on the fact that while syphilis was prevalent in India, aneurysm was extremely rare. It was an interesting fact that the patient in this case had, at the same time, two popliteal aneurysms and a swelling of the carotid at its bifurcation. Most surgeons would have hesitated to operate, and he would congratulate Mr. Spencer on the success of his intervention. He himself would not be keen on doing proximal ligature in such a case, but, if pressed, he would probably attempt it. He suggested, however, that it might be better to try the effects of distal ligature, followed by the injection of a coagulating fluid into the sac. Would some experimental physiologist say what coagulant could safely be introduced in the aneurysmal sac after the application of a proximal ligature ?

Dr. SEYMOUR TAYLOR, in reply, agreed with the last suggestion, adding that, of course, ligature of the common carotid presented two great risks, viz., sepsis, and the danger of hemiplegia as the result of interference with the cerebral circulation. If the obstruction to the circulation could be done gradually, it was quite possible that a collateral circulation would be established. He had not attempted any treatment so far, but as Mr. Spencer described it as syphilitic arteritis he would now try the effect of antisyphilitic medication.

CASE OF DIABETES INSIPIDUS.

By W. J. HADLEY, M.D.

THE patient, a woman, aged 22 years, had suffered from her present malady for the past two and a half years, and from dyspepsia and constipation for the last four years. There was no family history bearing on the case. Thirst and polyuria had been considerably worse of late, and she had lost 2 stone in weight in the last two years. She had had facial erysipelas on several occasions, as well as attacks of giddiness with temporary misty sight. There were no other signs of nervous disease. The average amount of urine passed in 10 days was 282 ounces per diem, whilst the average daily amount of fluid taken in the same

period was 216 ounces. The urine contained a faint trace of albumen, no sugar; and the urea, though relatively below normal, was really in excess.

Dr. Seymour Taylor said that on first seeing the patient he thought it was a case of myxœdema. Her face was swollen and bloated, and there was a tendency to the red patch on the cheeks. Her voice was of the typical leathery intonation, and he thought, if the case were watched, symptoms would develop showing that it was one of typical myxœdema. It was just one of the kind of cases on the borderland referred to by Dr. Ord in his last Bradshaw Lecture. The diagnosis, however, of the existence of diabetes insipidus was beyond dispute.

Dr. Robert Maguire said the pathology of these cases was extremely obscure. At one time it had been customary to refer all such obscure cases to the nervous system; at present the fashion was to attribute them to toxæmia. Here, however, he would suggest that probably the blood-tension was at fault. It was difficult to decide the exact state of the blood-tension in such cases, and neither the finger nor the sphygmograph gave trustworthy records. He had recently tried to use the sphygmometer, but with indifferent success. If, in the future, they could use some means of ascertaining and recording more accurately the habitual state of the blood-tension in general, they might obtain information of value. In this case the tension appeared to be higher than normal.

Mr. Battle recalled that when house-physician at St. Thomas's, under Dr. Payne, a young man was admitted unconscious, and passing many pints of urine in the day. At the *post-mortem* a gumma was found in the floor of the fourth ventricle. He asked if there was any specific history in the author's case.

Dr. Hadley, in reply, said that he had noticed the myxœdematous appearance, and he had, moreover, noticed the same thing in other cases of the same kind. He thought it might be due to the enormous amount and the rapid consumption of fluid drunk by the patient, resulting in heightened arterial tension, which in its turn might determine the bloated appearance. The voice was worse when she had been without drink for some time. He thought this peculiarity was due to the sticky dryness of the fauces. The blood-tension appeared to him to be medium. There had always been a faint but unmistakable trace of albumen in the urine, and in such cases there was always a doubt as to whether they might not be attributed to commencing granular kidney. The patient was a single woman, who had never had a child nor a miscarriage, and there was nothing to suggest a specific history. He had, therefore, not administered iodide of potassium. Bernard had shown that polyuria might be produced by puncture of the fourth ventricle at a point higher than the spot which, if punctured, gave rise to glycosuria.

FIVE CASES.

By W. H. Battle, F.R.C.S.

(1) *Case of Sarcoma (?) Treated by Coley's Fluid.*

Mr. Battle showed a man who had already been exhibited* before the Society while under treatment for sarcoma by Coley's fluid. The patient at that time presented a large tumour under the right clavicle extending down the chest; another, above and attached to the clavicle, extending into the posterior triangle of the neck; and others over the sternum, &c., with numerous enlarged glands in the axilla. He was treated by half-minim doses of Coley's fluid injected into the arm, and the doses were gradually increased to two minims. The total quantity did not exceed 24 minims. The treatment was carried on for three months, ceasing in May last. Progress had been very satisfactory, and now there was no actual trace of the tumours. Microscopical examination of portions of the growths from the sternum, &c., had shown them to be composed of spindle cells with some large cells. The patient's general health was quite good, the scars soft and non-adherent, and he was earning his living as a tram conductor. The only trace of former troubles was the dilated condition of the veins of the right upper arm. A radical cure for inguinal hernia was performed in July without any unusual incident attending it.

The President observed that this was a most important case, but the surgeon who introduced it, of course, did not expect to be allowed to leave the room without undergoing severe cross-examination. Cases of sarcoma treated by Coley's fluid were full of interest, but why did the author say so definitely it was a sarcoma? It appeared to him, with all respect to the microscopists, that there was nothing so likely to lead surgeons astray as the result of the examination of specimens removed from living subjects. He would require some better assurance than that before he could concede that it was a sarcoma. If it were so, why were the glands in the axilla enlarged? It would be very unlikely that a sarcoma of the breast or in its neighbourhood would infect the axillary glands. He suggested that it might be a case of granuloma of syphilitic or tuberculous origin. He asked whether the patient had had syphilis, and whether he had been treated by iodide of potassium? He knew of one case that had been treated for malignant disease of the end of the femur by Coley's fluid, and it eventually found its way into another

* 'Transactions of the Medical Society of London,' vol. xxi, p. 362.

hospital, where it was excised as an undoubted case of tuberculous hip-joint disease. If that case had got well under Coley's fluid, what a splendid triumph it would have been! He admitted that he was a sceptic, and that he wanted these cases proved up to the hilt before their being recorded.

Mr. Mansell Moullin congratulated the author on having shown the first microscopically-proved case of dispersion of sarcoma by Coley's fluid in England. He himself had seen the tumour and the various secondary growths, and he had also seen the microscopical sections, and he could not conceive that the tumour was anything but sarcoma. He said there was a great tendency to doubt Coley and Coley's statements. Last year he himself had brought forward two cases before the Society in which the tumours had disappeared under this treatment. They were intra-abdominal tumours, so that he was not in a position to affirm positively that they were sarcomatous. He had also published a third case of a tumour growing in the iliac region, which also did not admit of conclusive proof, but it completely disappeared. He admitted that none of his cases could be proved to be sarcomatous, but they were certainly not inflammatory or gummatous, and they disappeared under treatment by Coley's fluid. Mr. Battle's case was the first in which there had been a thorough microscopical examination. Coley had published a large series of cases of spindle-celled sarcoma, everyone of which had been favourably influenced by his fluid, though all had not been cured. In a large proportion of the cases the tumours had disappeared completely, and in some there had been no recurrence for upwards of seven years. These cases were authenticated by microscopical examination by well-known American observers. He believed that in Coley's fluid they had a remedy for spindle-celled sarcoma, not perhaps an ideal remedy, because it was somewhat dangerous and it did not succeed in all cases, but they were evidently on the right track. Recurrence sometimes took place, but so it did sometimes after operation, yet he had never heard operation decried because it did not afford a guarantee against recurrence. Therefore, if recurrence did take place with the fluid, the latter was not in a worse position than operation. He did not pretend that it ought to take the place of operation, but in cases where operation was no longer feasible, there was a very fair chance of a spindle-celled sarcoma disappearing without recurrence, if treated by Coley's fluid. He pointed out that few cases had been cured with the very small doses employed by the author, and in most of the cases there had been severe rigors, the absence of rigors in the author's case being probably unique.

Dr. Robert Maguire related a case at St. Mary's Hospital referred to him by Mr. Lane, who had amputated through the upper third of the thigh for sarcoma. The patient had spat blood, and on examination Dr. Maguire found evidence of recurrence in the lungs and also in the stump. It was an absolutely hopeless case, but, with the patient's consent, he tried Coley's fluid, injecting it directly into the veins of the arm so as to obtain a local as well as a constitutional effect on the lungs. He injected first three, and then five minims every other day for 10 days without the slightest ill-effect, and with relief to some of the symptoms. The patient died, and, *post-mortem*, it was seen that the case was necessarily quite hopeless, the lungs being riddled with sarcomatous nodules.

Mr. Waterhouse concurred with the President in being absolutely sceptical with regard to Coley's fluid. His own experience was limited to five cases of inoperable sarcoma. In three of these cases rigors occurred

and in two, even with medium doses, the patients had died of hyperpyrexia. It was, therefore, a most dangerous remedy, and in no case in his experience had it done any good whatever. As bearing upon the question of the diagnosis of sarcoma, he related the case of a German merchant suffering apparently from sarcoma of the lower third of the femur. The patient absolutely declined amputation, but he allowed an incision to be made into the mass from which specimens were removed for examination. One portion was sectioned for him, and it was declared to be a round-celled sarcoma, and the other portion was sent to a relative of the patient, a well-known pathologist in Germany. He also reported that it was sarcoma. Nevertheless, two or three months later some suppuration occurred, and a piece of necrosed bone was removed. This patient recovered without the aid of Coley's fluid.

Mr. Battle, in reply, recalled that the patient was shown last year whilst undergoing the treatment. His own impression at first was in favour of a syphilitic origin, and the man was treated by iodide of potassium in increasing doses until he was taking as much as 25 grains three times a day. This was continued for two months without producing any effect whatever on the tumours. In order to ascertain the exact nature of the growth, sections were taken from the tumour over the sternum, and also from over the clavicle. These were examined by Dr. Jenner and by Mr. Shattock, from both of whom he received the assurance that it was a case of spindle-celled sarcoma with some large cells. While under iodide of potassium, the tumours had been slowly getting larger, and the arm had become greatly swollen from pressure on the vessels. Having heard of Mr. Moullin's cases, he thought it worth while in the interest of the patient to try the effect of the treatment, especially as no operation could be thought of. The result they could see for themselves. The patient was now strong and healthy, and no trace remained of the tumours. He asserted his belief that it was a case of spindle-celled sarcoma which could not have been satisfactorily dealt with in any other way.

At the meeting of the Society held on November 28th, 1898, the President announced that in connection with the report of this case, one of the microscopists to whom the specimen had been entrusted had some doubts as to its sarcomatous nature. The question would therefore be referred to a sub-committee for examination, and report to the Society. Mr. A. Pearce Gould, Mr. Stanley Boyd, and Mr. A. Marmaduke Sheild were accordingly nominated to form the sub-committee.

At the meeting of the Society held on May 8th, 1899, the following report of the sub-committee was read by Mr. A. Pearce Gould :—

"*To the*

"*President of the Medical Society of London.*

"We have read the hospital notes of Mr. Battle's case, illustrating the advantage of Coley's fluid in the treatment of inoperable

tumours, and the published accounts of the case, and we have read Mr. Shattock's report on the microscopical appearances of the part removed, and have examined two microscopical specimens of the part removed, prepared by him.

"In our opinion—

"(1) The pathological appearances are not conclusive of sarcoma.

"(2) The clinical history does not exclude syphilis.

"(3) In view of the extreme difficulty of accurately diagnosing sarcoma from inflammatory formations, only those cases the nature of which has been indubitably proved should be admitted as evidence of the value of any therapeutic measure.

"(Signed) "A. Pearce Gould.
"Marmaduke Sheild.
"*May 5th*, 1899." "Stanley Boyd.

(2) *Case of Hepatoproptosis.*

Mr. Battle showed a woman, aged 64, who exhibited in a remarkable degree the phenomenon of hepatoproptosis. When she stood erect the liver came down into the iliac fossa, and, even when recumbent, the hand could be passed above it under the ribs. She had a lax abdominal wall, but did not suffer much from pain since she had been supplied with an abdominal belt. She ascribed her condition to her last confinement 25 years ago. The liver appeared normal in size and structure. She had not laced tightly.

(3) *A Man with Three Patellæ.*

Mr. Battle showed a man with three patellæ. A skiagraph of the third proved it to be formed mainly of bone. The normal patellæ were in their usual position, and were of the same size. The third or abnormal one was in the quadriceps extensor on the right side.

(4) *Case of Genu Retrorsum.*

Mr. Battle showed a lad exhibiting this curious and rare deformity. He was 15 years of age, and when he stood up the knee projected backwards. The weakness of the joint only dated

back five weeks, but was probably due to some defect of development of the upper epiphysis of the tibia. He proposed to fit the lad with a special knee-cap.

(5) *Case of Leontiasis.*

Mr. BATTLE also showed a patient with multiple fibrous bodies all over the skin, especially of the trunk and head. On the head the growths were soft and nævoid-like, whilst on the body they varied from minute pigmented points, with tumours as large as the head of a pin, to soft sessile projections the size of horse beans. One over the left breast was large, and the skin fell in folds, and was somewhat pigmented. The boy was of deficient intellect, and his body emitted an objectionable odour.

February 13th, 1899.

TWO CASES OF COXA VARA.

By W. WATSON CHEYNE, F.R.C.S., F.R.S.

Case I.—Showing Result of Operation six years previously.

A BOY, aged $9\frac{1}{2}$. First seen six and a half years ago, at age of 3, with deformity clearly referable to curvature of the neck of the femur, and chiefly characterised by marked eversion of both legs and greatly diminished power of inversion. Trochanters at level of Nélaton's line. On January 23rd, 1893, the following operation was performed on one side:—An incision on the outer side of the thigh at upper part down to the bone, femur sawn transversely below trochanters, foot and leg forcibly inverted till the normal degree of complete inversion was obtained, and, being held in that position, an oblong perforated aluminium plate was applied over the outer side of the femur at the point of division of the bones and nailed over the two fragments with nickelled tin tacks so as to keep up the inversion. Wound healed by first intention. Case shown at Clinical Society on November 24th, 1893 (*see* 'Clin. Soc. Trans.,' vol. 27), under title of "External rotation of both legs." (The name "coxa vara" was not introduced till 1894, and hence this case has escaped notice in the literature of that subject.)

On January 7th, 1896, the boy was admitted to Paddington Green Children's Hospital with an abscess at the seat of the former operation, on opening which the plate and tacks were found loose, and were removed.

Case shown to-night for three reasons—

(1) The object of the operation has been completely attained; the leg operated on is in perfect position, and as useful to him as if there had never been anything the matter with it.

(2) The other leg, which was useless before the operation, has improved so much in usefulness that the parents have never seen the necessity for any operation on it; nevertheless, the typical deformities of coxa vara are present, viz., marked external rotation, imperfect power of internal rotation, and elevation of the trochanter above Nélaton's line. Some improvement has, however, taken place in the way of internal rotation, probably as the result of the swinging forward of the leg in order to bring it in a line with the other.

(3) A result entirely unexpected by me is shown, viz., that, as a consequence of the operation, *the deformity of the neck of the femur has been arrested.* The trochanter on the side operated on still remains at the level of Nélaton's line, as it was at the time of the operation, while on the other side it is more than half an inch above it, and there has been no increase in the backward curve.

How may this be explained? Two factors are conceivable. (*a*) The processes of repair and consolidation at the seat of fracture may have extended into the neck of the femur and led to consolidation there and arrest of the softening process which was leading to the curvature. (*b*) The branches of the nutrient artery running upwards to the neck of the bone were, of course, divided in the operation, and, possibly as a result of diminished vascularity, the consolidation of the neck may have been hastened.

Case II.—Showing Result of Operation two years previously.

A girl, aged 5; had been more than once in Paddington Green Children's Hospital for incipient rickets. Admitted for operation in January, 1897, when 3 years of age. At that time there were all the signs of coxa vara in both legs; marked eversion of legs, inversion even to right angle impossible, difficulty in abduction,

lordosis. The trochanters were at the level of Nélaton's line, anterior curvature of the femora, flat feet; the child walked with a waddling gait, and the knees tended to cross one another.

On January 26th, 1897, the right femur was divided below the trochanter, the lower fragment inverted and the bones fastened together with an aluminium plate as in Case I. Union by first intention.

Results are the same as in Case I:—(1) A good leg on the side operated on. (2) Improvement in the usefulness of the left leg. (3) Arrest of the deformity of the neck of the femur on the side operated on, while it has increased as regards elevation of the trochanter on the other side. The left trochanter is now about half an inch above Nélaton's line.

The aluminium plate is still present in the leg.

Mr. Jackson Clarke said that he had seen the little girl before the operation, and he thought the improvement was marked as compared with her then helpless condition; her posture and gait were ludicrous at that time, but they had now become physiologically adequate. He had had four of these cases during the last year or two, and was enabled to show a photograph of one of them exhibiting complete eversion of the feet. There was half an inch elevation above Nélaton's line. Three of these children were 2, 2, and 3 years of age respectively. In these cases, as rickets was in active progress, he had instruments made that kept the feet turned forward at the maximum of inversion, allowed the patient to walk, and took the weight off the hip-joint, transferring it to the ischia by perineal bands. The conditions, in two of the cases, had greatly improved in a year; so much so, that no operation was thought necessary. In the third case, which had only been under treatment for three months, similar improvement was taking place. In the fourth case, he had diagnosed an incipient stage of this affection. There was a great deal of pain in the hip-joint, some of it referred to the knee-joint simulating an early tuberculous coxitis. In that case, after a period of extension and the use of the apparatus, the symptoms entirely subsided so that there was no deformity. He took this to be an early stage, the stage preceding actual deformity—a stage which he thought should be looked for and treated more than it is.

Mr. Watson Cheyne, in reply, said he had not overlooked the question of splints in these cases, but he had not been able to devise anything likely to answer the purpose he had in view. He mentioned that of late another operation had been proposed and carried out in adults, viz., the removal of a wedge from the neck of the femur. Theoretically, it was a much more perfect operation than one which left the neck uncorrected, but in a little child with a small neck to the femur and little to work upon, together with the great difficulty of retaining the parts in proper position afterwards, he thought it was a much better plan to treat it by division of bone. At the same time he was not at all sure that, in the adult, the removal of a wedge would not be the best course.

CASE OF RECOVERY AFTER OPERATION FOR CEREBELLAR ABSCESS.

By Robert Maguire, M.D., F.R.C.P.

The patient was first seen in private consultation on March 2nd, 1896. He had suffered from discharge from the right ear for some time, and suddenly, as the result of a chill, the discharge ceased. The patient complained of pain in the right side of the head, behind the ear. He became rapidly comatose, previously showing convulsive movements of the left side. When first seen he was comatose, the head turned slightly to the right, the left arm and leg flaccid, the mouth turned slightly to the right, and the right pupil larger than the left. He could be roused sufficiently to give a stupid monosyllabic answer. The temperature was then 101°. Operation with the existing surroundings being impossible, he was removed to St. Mary's Hospital in the afternoon of the same day, and on arrival showed, in addition to the symptoms mentioned, distinct weakness of the right arm and leg. On March 3rd, Mr. Pepper operated by trephining behind the right ear. The dura mater and pia mater, and also the superficial cerebellar substance, seemed normal, but on cutting through about a quarter of an inch of cerebellar substance, an abscess was reached and about 2 ounces of pus were evacuated. Next day the patient was conscious, and answered questions rationally. There was still a little weakness of the left side, the right side being normal. The temperature in two days more fell to normal. Convalescence was uninterrupted, and the patient was discharged cured on April 20th, 1896. He has had no bad symptoms since then.

Dr. St. Clair Thomson asked whether there was any reason why the patient's mastoid antrum was not opened at first, for, although infections could pass up through the roof of the tympanic cavity, he did not think they could travel backwards towards the lateral sinus and the cerebellum without passing by way of the mastoid. If that were so, this patient's mastoid was still infective, and might expose him to further trouble. When these patients presented themselves without a history, the presence of a fœtid discharge would attract attention to the side affected. He asked whether the trephined bone was replaced, remarking, at the same time, on the firmness of the cicatrix.

Dr. Ormerod said he was not clear as to whether the original paralysis was on the right or on the left side.

Dr. Maguire, in reply to Dr. Ormerod, said that the paralysis was on

the left side. He remarked that in the first place he had made up his mind that there was an abscess of the cerebellum, and that it was not superficial. It was greatly to Mr. Pepper's credit that, without any history, he also had arrived at the conclusion that this was the case. On consultation, they thought it would be better to go direct to the abscess without halting midway. He was surprised that the membrane showed no change, but they were so convinced of their diagnosis that they went on. He agreed that the patient ought to have some advice about his ear, as there was still a discharge. He thought the bone had not been replaced.

PHARYNGO-MYCOSIS LEPTOTHRICIA, BUT PROBABLY MORE CORRECTLY CALLED KERATOSIS PHARYNGIS.

By St. Clair Thomson, M.D.

The patient—a man, aged 38—shows on the faucial and lingual tonsils the characteristic appearances of what is generally termed pharyngo-mycosis leptothricia. The crypts of the tonsils are occupied by small, isolated, tough, firmly adherent, white excrescences, which do not coalesce, and the mucous membrane beneath and between them is apparently healthy. These white masses sometimes project above the mucous surface in the form of tufts, brushes, or stalactites. They are very adherent, and generally break off short on being seized with forceps.

The affection causes no general symptoms, and, as a rule, no local trouble except some feeling of discomfort or irritation.

The outgrowths recur rapidly on being removed, and the affection may be very persistent. If left untreated it generally disappears and leaves no trace behind; but it is said to have lasted for five, and even eight years.

It is generally found in persons in the prime of life, and is said to be more frequent in females.

It is limited in distribution by the boundaries of Waldeyer's ring, and this situation, together with the appearances and the absence of local reaction and general symptoms, serves to distinguish it from lacunar tonsilitis and diphtheria. The diagnosis is important.

It has generally been attributed to the growth of the mycosis leptothricia. But the spores and even the mycelium thread of this fungus are nearly always present in the mouth and pharynx.

The attempts of Brown Kelly to inoculate it in his own throat always failed. So it is probable that the mycotic element is of secondary importance, and that the fundamental pathological condition is a cornification of the epithelium lining the tonsillar crypts—a condition for which Siebenmann proposed the term hyperkeratosis lacunaris.

The author has found no local application of any avail—not even a 10 per cent. solution of salicylic acid in alcohol. Change of air will frequently effect a cure. Local surgical measures with the forceps, sharp spoon, and galvano-cautery may be relied on to effect a prompt and lasting cure.

Mr. Watson Cheyne asked for further information as to the microscopical characters, and asked what else was found beyond the leptothrix. He suggested that the appearances might be due to secretion from the crypts of the tonsils of a material which was a good pabulum for the organisms, and that thus it was a general rather than a local condition. He suggested removing the tonsils altogether.

Dr. de Havilland Hall said he had seen many of these cases under treatment, and agreed that they were very difficult to cure. He, too, had observed that, except in the case of voice users, the condition caused little inconvenience to the patient. He had at present under observation a barrister who complained very much of the trouble it caused him in his daily work. This patient had undergone much treatment before coming to see him. He started with a 15 per cent. alcoholic solution of salicylic acid, and had been much pleased with the result. The patient's throat was far too extensively affected to be attacked with the galvano-cautery, the growths extending right down into the larynx. It certainly was a most painful remedy when applied so low down. He thought, therefore, it should be cured without recourse to the galvano-cautery. His experience had led him to hope that they might be able to dispense with the cautery scraping and other surgical measures, except in cases which resisted this treatment.

Dr. St. Clair Thomson, in reply, said he was encouraged by Dr. Hall's experience to try a little longer with an increased strength of salicylic acid. He would try it on one tonsil, and keep the other for comparison. He agreed with Mr. Watson Cheyne that the pathology of this disease was in an unsatisfactory state. He thought the leptothrix must be given up altogether, but he did not think that calling it hyperkeratosis lacunaris would help them much. The pathology of pharyngeal affections presented some difficulty, as many organisms were present even in health, and much that one saw under the microscope could only be described as *débris*. He agreed that the general health must be investigated much more than was usually done. It was a common experience that if they could afford a change of air, such as a short voyage, these cases did well.

A CASE OF VERY LARGE THORACIC ANEURYSM.

By Hector Mackenzie, M.D., F.R.C.P.

Alfred T., æt. 44, came under treatment at St. Thomas's Hospital over two years ago for pain in the front of the chest and through the lungs to the left side, of a burning sharp nature. At that time there was no visible aneurysmal tumour, but there was dulness and pulsation in the second right intercostal space close to the sternum. The patient soon discontinued attendance at the hospital and followed his ordinary occupation as a labourer. Three months ago he returned with a large pulsating swelling in the upper part of the chest so large as to be quite conspicuous even without undressing. The tumour now measures 7 inches transversely and $5\frac{1}{2}$ inches vertically, while it projects $2\frac{1}{2}$ inches above the surface of the chest wall. Above, it reaches nearly to the clavicle; below, to the upper border of the fifth rib: to the right it extends to the nipple line; and to the left about an inch external to the sternum. The swelling is soft. The greater part of the gladiolus and the attached cartilages on the right side have been absorbed. On palpation there is a very diffuse and heaving systolic impulse, followed by a diastolic shock. No murmur audible. The tumour has considerably increased in size during the three months the patient has been under observation.

The case is shown on account of the exceptional size of the tumour and the comparative insignificance of the symptoms to which it gives rise. The pain is now very much less than it was two years ago. The patient does not feel ill enough to come into the hospital, but he finds the shortness of breath and pain produced by exertion rather trying.

Dr. de Havilland Hall said it was the largest aneurysm he had ever seen, and the patient was undoubtedly in a very dangerous condition. A patient now under his care at Westminster Hospital had an aneurysm that was undergoing very rapid development, and he had been admitted in order to undergo a modified course of Tufnell's treatment in the hope that fibrin might be deposited in the sac. Unfortunately neither the diet nor the iodide seemed to suit the patient, whose pulse rate went up and down and whose temperature rose. The treatment was, therefore, discontinued for some weeks, and the aneurysm rapidly advanced. In considering what could be done, he had dismissed electro-puncture as well as the idea of passing in fine steel wire. Then he thought of a series of cases reported by Lancereaux, of Paris, in 'La Semaine Medicale

treated by the injection into the connective tissue of the gluteal region of a 1 per cent. solution of gelatine in a 1 per mil. solution of chloride of sodium. Doses of 50 c.c., gradually increased to 250 c.c., were injected at the temperature of the blood. This treatment was commenced five or six weeks ago and continued up to the present with practically no discomfort, apart from the actual puncture and the consequent distension of the tissues. It was attended with very little risk, and if it held out a reasonable prospect of success, he thought it ought to be tried in these otherwise almost hopeless cases. He had made five injections, and had arrived at doses of 150 c.c., which he would increase next week to 200 c.c. and the week after to 250 c.c. Then, having reached the maximum, he would wait a while and see if the aneurysm showed signs of retroceding. Dr. Hall promised to report the further progress of the case later on.

Dr. Robert Maguire thought the patient was to be congratulated upon his determination not to go into a hospital for treatment. He asked whether anyone had ever seen a well-marked case of thoracic aneurysm in a working man from which recovery had taken place to such an extent as to enable him to resume work. This man had been earning a living for the last two years by working at the dockside, which was no sinecure. Another disadvantage of active treatment was that by placing the man on his back in bed and keeping him from moving, there was more pressure on the œsophagus, and ultimately, very often the aneurysm burst into the œsophagus. If one had the courage of one's opinions, one would urge such patients to go on working until the end came.

Dr. Hector Mackenzie, in reply, agreed that the man was probably better at work. At present he was in fairly good health and had suffered very little inconvenience, and that was why he had not been strongly urged to go into hospital. He had no knowledge of Lancereaux's treatment, and certainly would not expect it to give very encouraging results. When they had Dr. Hall's further report, that might decide him to adopt it in such cases.

CASE OF GRAVES'S DISEASE IN A MALE.

By Hector Mackenzie, M.D., F.R.C.P.

The patient, Daniel F., aged 32, station-master, is suffering from Graves's disease in a typical form, and shows all the usual symptoms. He was admitted to St. Thomas's Hospital on January 17th, 1899. He has always been of a nervous temperament, and suffered from an attack of nervous depression in 1882. He has found his duties as station-master at a small country station particularly trying, and for some time past has felt that he was easily knocked up and became soon out of breath. About six months ago he noticed puffiness over the eyes, for which he consulted a doctor. In the beginning of December, 1898, he observed that his eyes were protruding, and the doctor who then

examined him called his attention to the fact that his throat was swollen, of which he was previously unaware.

The eyes are prominent. When the patient looks down there is marked lagging of the upper lid, and the sclerotic becomes exposed between the lid and the cornea. On admission, when the lids were closed they did not quite meet, but this is now less marked than it was a month ago.

The enlargement of the thyroid is considerable, soft, and symmetrical. The circumference of the neck is 15 inches. A loud blowing systolic murmur is audible over the goitre. The pulse rate has varied between 90 and 120. There is marked carotid pulsation in the neck.

Pulsation is also visible in the second, third, fourth, fifth, and sixth left intercostal spaces. On auscultation over the sternum and along the left edge there is a very rough systolic murmur, best heard in the situation of the third left costal cartilage. The murmur is also audible in the aortic area but not at the apex of the heart.

The skin is dark, but no patches of pigmentation are visible. The temperature has varied but little from normal. The highest temperature has been that recorded on admission, 99·8° F., the lowest 97°. The appetite has been good, and there has been no gastro-intestinal disturbance.

The urine has been normal in amount. No albumen present. On one occasion it was found to contain some substance which reduced Fehling's reagent, probably glycuronic acid.

The case is shown in the first place on account of the rarity of the disease in the male sex. In the last five years I have had six male cases under my care, but in the previous 10 years I saw only two cases in males. I estimate the comparative frequency of the disease in this country in the two sexes as about 1 to 20. It is possible that occupation and mode of life may act as exciting causes. Two of the other male patients, whose photographs I show, were, like the present subject, railway servants. It is stated that exophthalmic goitre is not infrequent in the Russian army.

Second, I wish to draw attention to the occasional presence in the urine in cases of this disease of some substance which is not glucose, but reduces Fehling's solution.

Third, I would allude to a very interesting symptom present in this patient which I have not previously observed, and I would

ask if it has been noticed by any Fellows of the Society present. This symptom is itching of the skin. In the present patient itching of the skin has been very troublesome for some months. The patient says he had "to scratch himself like a monkey." The interest of this symptom is that it is not uncommonly met with during the process of cure of myxœdema by means of thyroid preparation, and is often a cause of a considerable amount of suffering in these cases.

Mr. Watson Cheyne said he had operated upon a number of these cases, as advocated in Germany, on the supposition that the disease was due to hypersecretion of the thyroid. The tremor and palpitation quickly improved after operation. In one case, the patient, a woman, was soon able to do her sewing. One man was particularly pleased because he was able to play "Nap." within a few weeks of the operation. He could not say how long the improvement lasted; some patients had come back after a year or two saying they were still much better. He had, however, never seen the slightest benefit as regards the protrusion of the eyeballs.

Dr. Robert Maguire said it was perhaps natural that he, as a physician, should rely more upon drugs than upon surgical measures. He called attention to the use of Cactus Grandiflora in exophthalmic goitre and nervous palpitation. He had used it without a single failure for the relief of the palpitation, but no effect was produced upon the protrusion of the eyeballs. Sometimes all the other symptoms had disappeared, but never the exophthalmos.

Dr. Alexander Morison asked how long the patients had been under observation before improvement took place, and how long they had suffered from the disease. [Dr. Maguire said that two of the patients had been under observation for five or six years without much relief to their symptoms.] He thought it was generally admitted that many of these cases, by mere efflux of time, tended to improve. A patient of his own who had had syphilis, did not improve under anti-syphilitic treatment, and he saw no more of him for seven years. He had, however, recently learned that the condition had practically disappeared without any other treatment. He was, therefore, inclined to attribute the improvement to the subsidence of the condition which underlay the disease. He regarded the hypersecretion of the thyroid as a complication and not as the essential feature.

Dr. Hadley observed that some years ago inquiry began as to what became of these patients, and it was found that many of them returned in after years, having lost one or more of the symptoms—palpitation included—but that most of them retained the exophthalmos. The enlargement of the thyroid sometimes had disappeared, but not always. A few cases drifted into lunatic asylums and a few were seen *post-mortem*. He had seen two of these cases *post-mortem*, death having occurred in one from granular kidney, and in the other from endocarditis.

Dr. Hector Mackenzie, in reply, admitted that he had not tried Cactus Grandiflora. Several of his patients had been operated upon by removing half the gland, but he could not say that, on the whole, they had done any better than others under observation for an equal length of time. He had looked up the statistics of operation and found that the

mortality was by no means inconsiderable, being something like 12 per cent. directly from the operation. He endorsed the view that the tendency of the disease was towards limited recovery. He had seen few cases of complete recovery, but a considerable number had recovered sufficiently to show that the disease was only present in a very slight form. He had told many of his patients to discontinue treatment, and to see him at intervals of a year or two, and he found that quite a large number had done well. He also referred to the case of a patient who had had the disease for 17 years without consulting anyone. Dr. Mackenzie prescribed for him and had then lost sight of the man, but subsequently heard that he had died of an acute attack of diarrhœa, to which such patients were particularly liable, as they all knew. The greatest danger to life occurred during the first 12 or 18 months of the onset of symptoms. He agreed that the mental element was prominent, and was, in many cases, very difficult to combat.

SUBCORACOID DISLOCATION OF HEAD OF LEFT HUMERUS, WITH FRACTURE OF THE GREAT TUBEROSITY.

By V. Warren Low, F.R.C.S.

The subject of this note is a healthy and sober man, of 51 years of age. On the evening of Sunday, October 30th of last year, he was hurrying down the stairs of Farringdon Street Station, when his feet slipped and he stumbled down some 15 or 20 steps, falling in a heap at the bottom, and striking his left shoulder.

He immediately picked himself up, and, thinking that he had merely sustained some bruising, used his left arm to straighten his hat, which had been crushed in his fall. So little did his injuries inconvenience him that he attempted to board a train just leaving the station, but was prevented. When the next train arrived he remembers using his left hand to open the carriage door, and he did not suspect that his shoulder had been seriously injured till he arrived home and, on taking off his clothes, noticed that there was very considerable swelling and bruising in the region of the joint, and also that the "point of his shoulder" (acromion process) was abnormally prominent.

He came to the hospital early the next morning, when it was found that the left shoulder was very considerably swollen and bruised, but that the head of the bone was absent from its normal position, and could be felt below and to the inner side of the coracoid process, the axis of the humerus being correspondingly altered; no signs of fracture could be detected, and an unsuccessful

attempt was made to reduce the dislocation. He was then admitted into the hospital, and a few hours later, under an anæsthetic, further attempts at reduction were essayed; by this time the joint region was even more swollen than on admission, and it was evident that there was a very extensive extravasation of blood both in and around the joint. During one of these attempts crepitus was obtained, and it was thought that the shaft of the bone moved independently of the head; consequently, a diagnosis was made of fracture of the surgical neck of the humerus, with dislocation of the head.

For the next fortnight the joint was too painful to allow of any examination to be made without an anæsthetic, and as our Röntgen ray apparatus was unfortunately at this time under repair, no further investigation was made, the man being kept in bed with his arm on a pillow while evaporating lotions were applied. As his arm was absolutely useless, Mr. Owen, on November 21st, cut down on to the head of the bone, using the ordinary anterior incision for excision of the shoulder-joint. There were quantities of soft blood-clot both around and inside the joint capsule, while the following conditions were also found to be present:—

1. Subcoracoid dislocation of the head of the humerus.
2. The great tuberosity had been completely torn from the humerus and broken into a number of small fragments.
3. A small piece of the lower and inner part of the glenoid fossa had been broken off.

There was, however, no fracture of the surgical neck of the bone.

As even with the head of the bone thoroughly exposed it was impossible, for some reason, to restore it to its normal position, Mr. Owen removed the head by sawing through the surgical neck, and closed the wound.

The man's recovery was uneventful. The wound healed by first intention, and he left the hospital on December 7th, with some slight movement between the humerus and scapula.

Mr. Watson Cheyne asked what was the difficulty in getting the head of the bone back. He would scarcely have anticipated this resistance.

Mr. Jackson Clarke recalled the case of an elderly woman who had had her shoulder out for five weeks before coming to hospital. Reduction under anæsthesia by manipulation was found impossible. There was

much œdema of the arm from pressure on the axillary vein. He cut down and put in a strong retractor, forcing the bone into position without opening the capsule, and it remained in the glenoid cavity about a fortnight, and the œdema disappeared. Then the œdema returned owing to the head of the bone having slipped out. It was easy to return the bone, but the œdema persisted and even increased. He excised the head of the bone, but still the œdema increased, and the arm became so useless that it had to be removed. Looking back at the case, he thought that if he had opened the capsule in the first instance, and had made a complete diagnosis, in all probability, as the external tuberosity was entire, he would have been tempted to wire it to the head of the bone. He remarked that the movements of the joints after excision were usually very restricted, and if one could obtain union of the great tuberosity the result would be better. Of course, if the tuberosity were comminuted, there was no other plan than excision. If they brought a number of these cases together and compared notes, they might be able to formulate a better plan of treatment.

Mr. Warren Low, in reply, admitted that he was unable to explain the difficulty in replacing the head of the bone, though several serious attempts were made. With part of the glenoid cavity fractured, and comminution of the tuberosity, it was considered that excision would leave a more useful limb. He thanked Mr. Clarke for bringing forward his account of a similar case.

April 10*th*, 1899.

A CASE OF TRAUMATIC CEPHALHYDROCELE.

By W. H. Battle, F.R.C.S.

The patient, an infant 3 months of age, had a swelling on the right side of the head which was soft, fluctuating, and pulsated. Everted edges of bone could be felt along the margin of the swelling. Mr. Battle thought the site was too much in front of the fronto-parietal suture to be due to imperfect closure, and was disposed to attribute it to injuries received at birth. The child was the ninth, and forceps were used. The swelling was noticed three weeks after birth. He observed that the case was interesting on account of its early supervention. The only treatment had been a bandage and a pad over the part. The swelling measured 2 inches from above downwards, and $1\frac{1}{4}$ inches from behind forwards. There were no symptoms even when the swelling was partly reduced by pressure.

A CASE IN WHICH RECURRENT MAMMARY CANCER DISAPPEARED AFTER TREATMENT BY OOPHORECTOMY AND THYROID EXTRACT.

By G. Ernest Herman, M.B., F.R.C.P., F.R.C.S.

In introducing this case Dr. Herman mentioned that a similar case reported by him in 'The Lancet' of June 11th, 1898, had had no sign of recurrence, and that the patient had gained about 5 lbs. in weight since that report was written.

The patient whom Dr. Herman now showed had had three children and two miscarriages, the last pregnancy being 14 years ago. She first noticed a lump in her right breast towards the end of 1894, she being then aged 45 years. In May, 1895, she went to the Middlesex Hospital, and on June 1st Mr. George Lawson removed the right breast with the fascia covering the pectoral muscle, and four enlarged axillary glands. The growths on microscopical examination proved to be carcinoma. On October 21st of the same year she was readmitted with three nodules situated just beneath the scar, and just external to the anterior axillary line, and two others just above the centre of the scar. There were also further lumps in the axilla, which were thought to be enlarged glands. The nodules and enlarged glands were removed on October 23rd. (For these facts Dr. Herman had to thank Mr. Sydney Hulke, surgical registrar to the Middlesex Hospital.) The patient stated that "the old wound reopened" in April, 1896, and from that time continued to discharge. In November, 1897, she noticed a lump in the left breast. She was brought to Dr. Herman by Mr. Elliott Browne in July, 1898, and was admitted into the London Hospital on July 11th. There was then an ulcerated surface over the third and fourth ribs, depressed at its centre, and fixed to the subjacent parts, with thickened margins. Its general shape was that of an oblong with spurs projecting here and there, the dimensions of the oblong being $3\frac{1}{2}$ inches by $1\frac{1}{2}$ inches. In the right axilla was a depressed cicatrix fixed to the rib, separate from the ulceration, and without sign of recurrence of cancer. In the left breast was a lump of stony hardness which measured with callipers (the skin over it being therefore included) $3\frac{1}{2}$ inches by 2 inches. The left nipple was

drawn in. There were hard, enlarged glands in the left axilla. The patient had menstruated regularly until six months before admission. Since then menstruation had been irregular, the last time being three months before admission.

On July 12th both ovaries were removed, and as soon as the vomiting caused by the anæsthetic had subsided the patient was put upon thyroid extract, 5 grains three times daily. This she has taken ever since. She left the hospital on August 5th. Her weight was then 12 st. $2\frac{1}{2}$ lbs. She came to report herself on September 15th. The ulcer had completely healed. The left breast was much softer. Her weight was 13 st. $7\frac{1}{2}$ lbs. She was next seen on October 14th. The scar in the right axilla was movable. There was no distinct lump in the left breast and there were no enlarged glands in the left axilla. On March 28th, 1899, her weight was 14 st. 10 lbs. There were some keloid-like nodules in the scar, but these had not grown, and showed no tendency to break down. The left breast presented the characters of health, except that the nipple was retracted. Her right arm and hand swelled occasionally after scrubbing, but this had been the case since the first operation.

Mr. Stanley Boyd congratulated Dr. Herman on the remarkable success of his treatment. He was, however, sorry to be unable to agree that the nodules round about the scar were keloid. He thought those little red blotches also, with slight thickening, were really malignant, and would sooner or later develop. He had seen exactly the same lesions in his second published case—that of a woman, aged 35, the side of whose chest was covered with secondary nodules. All disappeared, and at first sight the surface seemed normal; but on close observation one saw small red patches, such as these in the present patient, which increased considerably in redness on handling, and each quickly redeveloped as a cancer plaque. Mr. Boyd was consequently afraid to take so optimistic a view as Dr. Herman in the case of the patient exhibited. Mr. Boyd raised the question as to what it was in the treatment that produced this marked and most beneficial effect. The author had quoted his cases in which thyroid was not given, and had stated that the results obtained were not so good as were those in the few recorded cases in which thyroid had been combined with oophorectomy. He himself could not agree with this conclusion, and he thought that if these two modes of treatment were always employed together, their respective effects would never be known. Thyroid was said to be a "powerful lymphatic stimulant," but what did that mean? Because the exudation in myxœdema disappeared when thyroid was given, could we assume that anything else lying in lymphatics would be similarly absorbed? He had not been able to learn from Dr. Beatson any, to him, satisfactory reason for thinking that the thyroid had anything to do with the success he, Dr. Beatson, had achieved. First of all, Dr. Beatson put

his patients on thyroid and nothing happened; then, after six weeks, he did oophorectomy and continued the thyroid, and improvement resulted in certain cases. Now, in two cases Mr. Boyd had obtained almost complete disappearance of all cancer without any thyroid at all, and one of these was well, two years having elapsed. He thought it had not been demonstrated that the addition of thyroid would improve upon this result, and he believed that Dr. Herman's other successes had been due to the oophorectomy and not to the thyroid. To resort to thyroid when the influence of oophorectomy had ceased was, he admitted, to put it to a severe test, yet the amount of disease to be dealt with was often less than when the oophorectomy was done. He referred to the case of Bishop and Page, which was operated upon, recurred twice, and was then regarded as hopeless. She was put on thyroid, and for twelve months no improvement whatever seems to have taken place; but some fresh nodules made their appearance. She was lost sight of for six months but continued taking thyroid, and he should imagine from the account that nobody was more surprised than Dr. Bishop to find then that the disease had altogether disappeared. He urged that the account given did not read like that of the steady action of a drug. But how else could it be explained? They all knew of a very remarkable case shown by Mr. Pearce Gould at the Clinical Society in 1897—that of a woman in the Cancer Ward in the Middlesex Hospital with absolute orthopnœa, one side of the chest being filled with fluid, and one thigh being helpless, shortened, and curved out in a most marked manner. She had also recurrent nodules about a breast scar. They thought she was dying, and no treatment was attempted; yet in a month the woman was much better, though she had taken nothing which could in any way account for the subsidence. When shown at the Clinical Society a few months later all signs of cancer had gone, her health had improved to a remarkable extent, and, though lame, she could walk without the aid of a stick. He had heard from Mr. Gould quite recently (March, 1899) that she was as well as ever. This was a case of undoubted cancer; there had been no treatment; and yet it had disappeared as completely as in any of the above referred to cases. Mr. Boyd commented on one fact in connection with this case, viz., that menstruation had ceased at about the time the improvement had begun. That, however, had often happened without any change in the growth of a cancer. The case of Bishop and Page was, perhaps, a parallel one to this. Mr. Boyd thought it was impossible, at present, to give a trustworthy opinion to a woman with cancer, who asks whether or not oophorectomy should be done—the results had varied so much under apparently similar circumstances. He had done the operation, in one case, in which the woman had ceased to menstruate only six months before; but, with the exception of the tendency to heal of an ulcer over the sternum, no improvement took place. It had seemed likely that operation after the menopause was not of much value; yet here was Dr. Herman's patient —irregular, not having menstruated for three months, and apparently close upon the menopause—proving a most brilliant success!

Mr. Battle thought that many of them would agree with Mr. Boyd that the patient was not cured. He himself thought that the nodules looked more like scirrhus than keloid. The left breast was small in proportion to the general development of the patient, and had a hard fibrous feel which gave one the impression that it was not normal. In some directions, it even suggested that there was a heaping up of fibrous

tissue almost amounting to a tumour. The patient's general condition was certainly wonderful, and he admitted that he knew no method of treatment which would produce that healthy appearance in a patient when she still had symptoms which previous experience taught them to regard as probably the remains of malignant disease. He hoped the author would let them know, on some future occasion, the ultimate result of the case.

Mr. G. R. Turner (Vice-President in the chair) thought the case was one of very great interest, and pointed out that even if some doubt remained as to the possibly scirrhous nature of these little nodules, there was the fact that a large cancerous open ulcer had healed leaving only these doubtful nodules. That alone was satisfactory. Then, again, the left breast, though there was some suspicion of hardness, had, they must remember, contained a large tumour measuring 3 inches across. Even if the improvement were only temporary, it was none the less very remarkable. Moreover, he would not diagnose, with absolute certainty, those nodules as scirrhus. It had been his fate on several occasions to remove such little nodules only to find that they were not scirrhus as he had feared. Again, even if actually carcinomatous, these nodules might ultimately disappear. He concluded by saying that he should certainly recommend the treatment for trial by the combined method, as the thyroid could do no harm even if it did not do the good suggested.

Dr. Herman, in reply, said that he could not agree with Mr. Stanley Boyd in thinking that oophorectomy alone would explain the benefit. Mr. Stanley Boyd had published five cases in which thyroid extract was given. In one it was only given for a month ; in another it was not begun till oophorectomy had failed and recurrence had taken place ; in three other cases he gave thyroid extract to patients whose ovaries had not been removed, and in two of them some benefit seemed to follow. Page and Bishop have published a case in which cure followed administration of thyroid extract. Four cases have now been published of cancer treated by oophorectomy plus thyroid extract, and in three of them the cancer disappeared. Dr. Herman submitted that the evidence at present before the profession goes to show that greater benefit results from the combination of oophorectomy with thyroid extract than from either of them separately, and that it is desirable that the effects on cancer of the combination of oophorectomy with thyroid extract should be tested as fully as possible. Then, as to the word "cure," no one could say that these patients had been in the strictest sense of the word "cured" until they had been watched through the remainder of their lives. But he submitted that to make cancer disappear was a much greater thing than to mechanically remove it with a knife, and if they could maintain that condition of the organism which led to the disappearance of already formed cancer tissue, they might reasonably expect that this condition would be incompatible with return of cancerous growths. These patients could not develop new ovaries, and they had been instructed to take thyroid extract for the rest of their lives. If they did so, he thought there was a sure prospect of freedom from cancer. In conclusion, he asserted that oophorectomy plus thyroid extract had given results which had not been approached by any other treatment of recurrent mammary cancer.

CASE SHOWING RESULTS OF GASTROSTOMY BY ALBERT'S METHOD.

By W. H. Battle, F.R.C.S.

The patient, a married woman, had been sent to Mr. Battle by Dr. Bowen-Williams with symptoms of obstruction of the œsophagus of some months' duration. This had begun in October, 1898, increasing until she was unable in February, 1899, to swallow solids at all. She had been given iodide of potassium, and bougies had been passed with only temporary improvement. He did gastrostomy by Albert's method and the patient recovered without a bad symptom. She had since had no difficulty whatever in alimentation. He mentioned that when allowed to swallow in the ordinary manner, the food came out of the opening, but this was not the case when introduced through the fistula. She had gained considerably in weight. This was the second case in which he had done Albert's operation, and in both it had answered very well, both patients leaving the hospital within three weeks.

NON-UNION OF FRACTURED HUMERUS.

By George R. Turner, F.R.C.S.

Mr. G. R. Turner showed a man who had fractured his arm at sea four years before, and who had been treated in the ordinary way by splints. Non-union occurring, he underwent an operation of some kind, the result being equally unsatisfactory. When he came to the Greenwich Hospital he had an ununited fracture of the humerus. He was syphilitic but there was nothing else the matter with him. Mr. Turner wired the ends of the bones but no union followed, although there was abundant formation of callus. He operated once more, this time with success, and the man was enabled to resume his occupation as a sailor. Sometime later, as the result of a violent lurch, he again fractured the arm, and once again Mr. Turner wired the fragments, getting them, as the skiagram which he handed round showed, in good apposition.

This time however, there had been very little attempt at repair, and little or no callus, and the question remained what it would be best to do under the circumstances.

Mr. Battle asked whether, at the first operation, the bones were rounded off, and whether fibrous tissue or muscle was interposed. He recalled a similar case, that of a patient under his care with an ununited fracture of the lower end of the humerus, also with a strong syphilitic history. He had done two operations, using ivory pegs instead of wire. The treatment appeared to answer for a time, but union did not take place satisfactorily. After the second operation more attention was given to the anti-syphilitic treatment, and sound union followed.

Mr. G. R. Turner, in reply, said that at the first operation the bones were pointed and had fibrous tissue between them. He had given the man mercury and iodide of potassium before and after the operation. He, too, had seen cases of ununited fracture in which syphilis appeared to be the cause, notably a fracture of the femur in a syphilitic subject, who was a long time under treatment. Union in this case ultimately took place without any operation at all.

PACHYDERMATOCELE OF THE SCALP (MOLLUSCUM PENDULUM).

By Francis Jaffrey, F.R.C.S.

Mr. Jaffrey showed a man, aged 32, suffering from a pachydermatocele of the scalp behind the left ear. The patient said it had been there as long as he could remember, and that he had never suffered from injury or abscess in this situation. The growth was slowly increasing in size, and is interesting inasmuch as it might be mistaken for nævus in the early stages.

Mr. Turner said the case reminded him of one of molluscum fibrosum in the neck. On the surgeon endeavouring to dissect it away, he was confronted with considerable hæmorrhage, showing that it was largely a cavernous angioma, and the patient died. Mr. Turner also recalled a case where this affection extensively implicated the buttocks. He suggested it was quite possible that there was nævoid tissue in the case before them.

EPILEPTIFORM CONVULSIONS FOLLOWING INJURY TO THE HEAD.

By Francis Jaffrey, F.R.C.S.

Mr. Jaffrey also showed a young man who in July last fell from a ladder, and was admitted to hospital with concussion of the brain and other injuries. He remained in the hospital a month, and six months later was again brought in with concussion. Since then he has complained of not being able to get work on account of the fact that it took him from 3 to 30 seconds to answer a question. When spoken to he raised his left hand to his mouth and appeared to be convulsed before he could speak, not, however, losing consciousness. There was a slight depression on the left side of the head.

Mr. Turner said the patient was at present under his care at St. George's Hospital. He had found on cross-examination that the man had been an epileptic for a long time, his accident being the consequence of his epilepsy. He had stuttered, too, though not to the same extent as now. Mr. Turner attributed the symptoms to the shock following the accidents, and not to any injury to the brain. He had given the man bromide of potassium, and in the quiet of the hospital the patient could give his name with comparative ease. He thought there was a large nervous element in the case.

Mr. Jaffrey, in reply, agreed that the depression in the skull had probably nothing to do with the symptoms. He mentioned that the man's condition had been the subject of two unsuccessful attempts to obtain compensation.

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